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The University of Western Australia

Faculty of Medicine and Dentistry

Semester 2 – Normal Systems (Medicine) IMED1100

2nd Semester Units

<table>
<thead>
<tr>
<th>UNIT CODE</th>
<th>UNIT NAME</th>
<th>POINTS VALUE</th>
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<tr>
<td>IMED1100</td>
<td>Normal Systems</td>
<td>17</td>
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<tr>
<td>IMED1112</td>
<td>Foundations of Clinical Practice 112</td>
<td>7</td>
</tr>
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<td>SUB-TOTAL</td>
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Introduction

In the second semester you start to learn about the form and function of the major systems of the body starting with the Cardiovascular and Respiratory systems from the organ and whole-body level down to the level of the cell. This unit runs alongside the unit FCP 112.

Context

At all times you should bear in mind why you are learning, and hopefully understanding, this material – it is not to learn all the fine details of Anatomy and Physiology – it is to make you a better doctor. To this end, although emphasis is on normal function, clinical examples are used extensively to highlight the basic mechanisms and principles of homeostasis and the relationship between form and function. The cardiovascular and respiratory modules are delivered at the same time as closely related problem-based learning sessions in the unit Foundations of Clinical Practice.

You should therefore try to learn what is important for medicine – use your lectures, the assessments and the clinical examples in your textbooks as a guide. You will NOT get this material in the same detail in later years. It is a hard task, but while you are adapting to university life and study in year one, you have to learn and absorb this material. The next time you try to remember the lobes of the lung you may be trying to listen to the breathing of a crying, distressed and ill child.

Science and Medicine

You might assume all medical practice is based on scientific evidence. Surprisingly the concept of “evidence based medicine” is quite a recent idea and is still resisted in some quarters. There is much bad science carried out by doctors, who have little or no scientific training, and who then use it in their practice. Our aim is to produce a new generation of doctors who understand how we obtain the knowledge we have, that is, who understand the scientific basis of medicine. Due to the huge strides being made in biomedical research, increasingly difficult decisions have to be made by doctors and society in medical matters such as cloning, euthanasia and genetic testing. It is essential that doctors who may have a major role in these debates understand and can interpret the scientific evidence.
Module 1  Cardiovascular System  8 hrs per week in the first 8 weeks of semester
Module 2  Respiratory System  8 hrs per week in the last 5 weeks of semester

Objectives:
1. To describe the normal structure and function of the cardiovascular and respiratory systems.
   Why: A doctor needs to understand the normal function of the body to later recognize and understand when normal function goes wrong - when your patient has a pathology. Cardiovascular disease is the number one killer in Australia. Respiratory tract problems form one of the largest case loads for any general practitioner.

2. To explain and illustrate the scientific basis of medicine.
   Why: Medicine is a dynamic and ever changing discipline. These advances are presenting society and medicine with huge ethical and scientific dilemmas, at a time when scientific literacy is falling. It is important that a modern doctor understands the way discoveries are made and the limits to such knowledge so that they can lead intelligent debate. A doctor must understand the need for “lifelong” learning. If your GP is 50 he / she may have learnt their medicine in the 1970's – do you want to be treated with medicine 30 years out of date?

If you think you would like to do research later in your career you might wish to consider doing the BMedSci course – one of the “cheapest” degrees you can get – you take one year out – usually in your third or fourth year - to do a research project and write a small thesis, but you get an intense training in research methodology.

Supplementary Reading and Study
To get good marks in university, the lectures and laboratories are a good guide to the core content. If you wish to get more than average marks you will need to study on your own (“homework”, if you like, does not stop at school). You will need to build up your vocabulary and your writing skills. Good spelling and grammar are crucial to accurate communication; bad spelling WILL be marked down. If English is not your first language, the university offers many aids and courses to help you improve your English – do not wait until the exam to find out this is holding you back. Get into the habit of reading around the subject, watch out for stories in the media, read general magazines like New Scientist and Scientific American to hear about the latest advances. Subscribe to internet sources of medical information. You will have to learn the habit of “lifelong learning” to keep yourself abreast of the latest developments in medicine throughout your career. Above all else you need to lose the habit you developed during TEE of being “exam focussed” and become “learning focussed”.

There is only one question your lecturers do not want to hear, that is “WILL IT BE IN THE EXAM”!!!
Unit Co-ordinator
Dr Shane Maloney (Discipline of Physiology)
Phone 6488 3394
Email: shanem@cyllene.uwa.edu.au

Deputy (Anatomy) Unit Co-ordinator
Professor Stuart Bunt (School of Anatomy and Human Biology)
Phone: 6488 2983
Email: smbunt@anhb.uwa.edu.au

Module Co-ordinators

<table>
<thead>
<tr>
<th></th>
<th>For the Physiology component:</th>
<th>For the Anatomy component:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular System</td>
<td>Dr Shane Maloney</td>
<td>Professor Stuart Bunt</td>
</tr>
<tr>
<td></td>
<td>6488 3394</td>
<td>6488 2983</td>
</tr>
<tr>
<td></td>
<td><a href="mailto:shanem@cyllene.uwa.edu.au">shanem@cyllene.uwa.edu.au</a></td>
<td><a href="mailto:smbunt@anhb.uwa.edu.au">smbunt@anhb.uwa.edu.au</a></td>
</tr>
<tr>
<td>Respiratory System</td>
<td>Professor Howard Mitchell</td>
<td>Professor Stuart Bunt</td>
</tr>
<tr>
<td></td>
<td>6488 3314</td>
<td>6488 2983</td>
</tr>
<tr>
<td></td>
<td><a href="mailto:mitchell@cyllene.uwa.edu.au">mitchell@cyllene.uwa.edu.au</a></td>
<td><a href="mailto:smbunt@anhb.uwa.edu.au">smbunt@anhb.uwa.edu.au</a></td>
</tr>
</tbody>
</table>

WEB CT
The Internet is the main means of rapid communication in this unit. Students must log on to WebCT and get into the habit of checking for notices regularly. Discussion groups will be encouraged and marks will be posted regularly. The inevitable changes in the course that occur will be announced here. There will also be links to Lectopia and copies of the lecturers Powerpoint presentations where these are provided.


SEMESTER DATES AND OTHER IMPORTANT DATES

Second Semester: weeks 31 to 44

- Week 31: classes begin Monday 28th July
- Week 38: study break Monday 15th September
- Week 44: classes end Friday 31st October
- Week 45: study break Monday 3rd November
- Weeks 46/47: exams Monday 10th November onwards
- Week 48: Summer Vac Begins (Yay!) Mon 24th November
ASSESSMENT

Assessment is progressive (accrues during the year) and uses a variety of assessment methods to ensure examination is fair and tests many aspects of your performance. Assessment can guide your learning process and can play different roles:

- **Formative** assessment provides the opportunity for feedback to be given to you in a practice situation without the performance being recorded e.g. practice computer based questions.

- **Summative** assessment contributes to your final mark. Assessment for each module will comprise a combination of continuous assessment and a final end of semester examination. Continuous assessment will be through laboratory reports, marked worksheets, MCQ quizzes and assignments.

- A **Barrier** assessment means an exam you must pass to move on to the next component of the course. The end of semester examination is a barrier exam and will consist of a total of 4 hours of written examination and practical examination. Theory papers will be multi-format (MCQ, SAQ and essays). Laboratory examinations will consist of “stations” that students pass around, answering a question at each station before a timed beep indicates time to move to the next station. Questions may involve more than just simple identification of parts and can examine any aspect of the course. The pass mark for these modules is 50%.

If you are ill or for any other reason such as a death in your close family, you feel your examination and/or learning process has been compromised, please fill out a form for ‘special consideration’ that can be obtained from the faculty office. Documentary evidence such as a medical certificate may be required. Only then will we be able to take these exceptional circumstances into account when marking your work or offering deferred examination.

Supplementary exams may be awarded. This normally only occurs for those obtaining 45 - 50%. Those scoring below 45% are not normally offered supplementary exams. Students should consult the Faculty handbook for rules relating to assessment.

All attempts are made to make the supplementary exam similar to the original exam but this cannot be guaranteed. The regulations state that the supplementary exam should be "similar" to the original exam. However for obvious reasons the questions have to be different to the original exam and the supplementary exam may therefore be harder or easier than the original exam. Similarly because of the reduced number of students, an oral (sometimes known as a *viva voce*) exam may replace the normal “spot” laboratory based exam.

It is far better to pass the first exam than to ruin your Christmas by having a supplementary exam.
ASSESSMENT: CVS AND RESPIRATORY MODULES

<table>
<thead>
<tr>
<th>Component</th>
<th>Percentage</th>
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</thead>
<tbody>
<tr>
<td>Physiology worksheets and questions (continuous assessment)</td>
<td>10%</td>
</tr>
<tr>
<td>Anatomy weekly worksheets and laboratory attendance</td>
<td>5%</td>
</tr>
<tr>
<td>Integrated Mid-semester CVS practical exam</td>
<td>20%</td>
</tr>
<tr>
<td>Final Integrated Practical exam</td>
<td>25%</td>
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<tr>
<td>Final Integrated Written exam</td>
<td>40%</td>
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</table>

The pass mark is an average mark of 50%, but note the two final integrated exams are barriers.

NOTE: In some circumstances, to ensure fairness and confidentiality in exams that are taken in several groups, students from a first group may have to be held in one room while others are examined. If this proves necessary, details will be provided nearer the time of examination.

Calculators: In some of the practical and theory exams calculators will be permissible. Only approved calculators can be used in tests or exams. Calculators are deemed “approved” by being marked with a sticker from the Faculty of Life and Physical Sciences (1st floor, Physics Building). Take your calculator to that office for approval. Programmable calculators, graphics calculators and calculators that have the capability to communicate with other devices are not permitted.

Laboratories in Physiology

The laboratory components of the CVS and Respiratory modules are an important part of your learning. Failure to attend laboratory sessions or to hand in assignments will result in a zero mark for that laboratory. You must attend your allocated laboratory session. If you need to change your lab time, either permanently or temporarily, you must get the change approved BEFORE the lab. Because lab facilities are utilised optimally you will not be able to attend an unscheduled session without approval. Because the labs are only 2 hours you must arrive on time and be prepared to participate in the pre-lab discussion. Lateness will be penalised 5% of your mark for that laboratory per 5 minutes.

Each of your Physiology labs in the CVS module and one in the respiratory module include a worksheet or assignment which is to be handed in for assessment by 5 PM on the Monday following the laboratory. There is an additional respiratory worksheet to be handed in at the end of semester as advised. Assignments are to be placed in the boxes marked with your demonstrator's name on the ground floor of the Physiology Department. The worksheets form part of your continuous assessment and will be marked as follows:

3 Good
2 Satisfactory
1 Borderline
0 Unsatisfactory
If you are absent from a laboratory class you must present a doctor’s certificate to your demonstrator as soon as possible. If the absence is legitimate an average mark based on that of your other worksheets will be given. If your demonstrator is not contacted you will receive zero for that worksheet.

Late submission will result in penalties – down 1 mark for each day. Thus if worksheets are handed in 3 days late, a zero mark for that lab will be awarded.

**Laboratories in Anatomy**

| We are dependent on donations of bodies to run this course, any inappropriate behaviour or breaches of confidentiality will be severely dealt with and can result in exclusion from the course. |

Anatomy quizzes will be handed out towards the end of each laboratory and collected the next week for marking. This also forms a means of measuring attendance. Tests not submitted by the following lab will be excluded **without exception**. If you are ill or have to miss a laboratory you need to fill in a form at the faculty office, the faculty will then inform the Anatomy coordinator who will make due allowance for the missing test and you will be awarded the class average mark.

**Laboratories general (Anatomy and Physiology)**

**Attendance**

Attendance at laboratories is mandatory. Failure to complete the practical work may preclude a student from sitting the final examination. **Attendance at all lectures is also assumed and not being present at a lecture when announcements are made is not an acceptable excuse for ignorance.** Please also check WebCT regularly.

**Illness**

If you are unable to attend through illness or any other reason you must contact the faculty office and a valid Doctor’s certificate, not signed by a family member, must be presented to the faculty office as soon as practicable on the student’s return to university.

**Leave**

You may not take leave within term or exam time without prior approval. Students are to seek approval from the faculty in the first instance. The unit coordinator will be informed by the faculty office if approval is granted. You should contact the coordinator to make arrangements to “catch up”.

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IMED1100 – Normal Systems 2008
### Lectures

<table>
<thead>
<tr>
<th>Day</th>
<th>Time</th>
<th>Location</th>
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</thead>
<tbody>
<tr>
<td>Tuesday</td>
<td>2 pm</td>
<td>Ross Lecture Theatre (Physics Building)</td>
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<tr>
<td></td>
<td>3 pm</td>
<td>Ross Lecture Theatre (Physics Building)</td>
</tr>
<tr>
<td>Thursday</td>
<td>9 am</td>
<td>Clews Lecture Theatre (Physics Building)</td>
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<td></td>
<td>4 pm</td>
<td>Clews Lecture Theatre (Physics Building)</td>
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### Laboratories

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<thead>
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<th>Day</th>
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<th>Location</th>
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<tbody>
<tr>
<td>Wednesday</td>
<td>10 am – 11.45 am</td>
<td>Anatomy – Main Dissecting Room</td>
</tr>
<tr>
<td></td>
<td>12 pm – 1.45 pm</td>
<td>Anatomy – Main Dissecting Room</td>
</tr>
<tr>
<td>Friday</td>
<td>10 am – 11.45 am</td>
<td>Physiology Ground Floor Labs</td>
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<td>2 pm – 3.45 pm</td>
<td>Physiology Ground Floor Labs</td>
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## CARDIOVASCULAR SYSTEM

<table>
<thead>
<tr>
<th>Week Yr/Sem</th>
<th>Date</th>
<th>Time</th>
<th>Lecture</th>
<th>Lecturer</th>
<th>Anatomy Lab (Wed)</th>
<th>Physiology Lab (Fri)</th>
</tr>
</thead>
<tbody>
<tr>
<td>31 / 1</td>
<td>Tue July 29</td>
<td>2 pm</td>
<td>Physiological Control Mechanisms</td>
<td>Dr S Maloney</td>
<td>Introduction to the Laboratory and the course</td>
<td>No Lab</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 pm</td>
<td>Autonomic Nervous System</td>
<td>Dr S Maloney</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>9 am</td>
<td>Mediastinum</td>
<td>Prof S Bunt</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>4 pm</td>
<td>Heart Development</td>
<td>Dr S Maloney</td>
<td></td>
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<tr>
<td></td>
<td>Thur July 31</td>
<td></td>
<td></td>
<td>Dr S Maloney</td>
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<td></td>
<td></td>
<td></td>
<td>Prof S Bunt</td>
<td></td>
<td></td>
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<tr>
<td>32 / 2</td>
<td>Tue Aug 5</td>
<td>2 pm</td>
<td>Electricity in the Body</td>
<td>Dr R Fox</td>
<td>Mediastinum</td>
<td>Symbiosis autonomic nervous system</td>
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<td></td>
<td></td>
<td>3 pm</td>
<td>Electrical events in the heart - ECG</td>
<td>Dr S Maloney</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>9 am</td>
<td>Heart and Pericardium</td>
<td>Prof S Bunt</td>
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<td></td>
<td></td>
<td>4 pm</td>
<td>Pressure &amp; Flow</td>
<td>Dr R Fox</td>
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<td></td>
<td>Thur Aug 7</td>
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<tr>
<td>33 / 3</td>
<td>Tue Aug 12</td>
<td>2 pm</td>
<td>The Cardiac Pump I</td>
<td>Dr S Maloney</td>
<td>The heart</td>
<td>ECG, Pulse Waves and Heart Sounds</td>
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<td>3 pm</td>
<td>The Cardiac Pump II</td>
<td>Dr S Maloney</td>
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<tr>
<td></td>
<td></td>
<td>9 am</td>
<td>Pressure Flow Relationships</td>
<td>Dr R Fox</td>
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<td>4 pm</td>
<td>Regulation of Blood Pressure</td>
<td>Dr S Maloney</td>
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<td>Thur Aug 14</td>
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<tr>
<td>34 / 4</td>
<td>Tue Aug 19</td>
<td>2 pm</td>
<td>Structure &amp; function in the Vascular System</td>
<td>Dr G Meyer</td>
<td>Histology of the cardiovascular system</td>
<td>Cardiac function</td>
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<td>3 pm</td>
<td>Arterial Stenosis</td>
<td>Dr R Fox</td>
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<tr>
<td></td>
<td></td>
<td>9 am</td>
<td>Patterns of blood vessels and lymphatics</td>
<td>Prof S Bunt</td>
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<tr>
<td></td>
<td></td>
<td>4 pm</td>
<td>Peripheral Circulation &amp; Regional Flow</td>
<td>Dr S Maloney</td>
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<td>Thur Aug 21</td>
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<tr>
<td>35 / 5</td>
<td>Tue Aug 26</td>
<td>2 pm</td>
<td>Capillaries, Veins &amp; Lymphatics</td>
<td>Dr S Maloney</td>
<td>Blood vessels of the torso</td>
<td>CVS changes in exercise</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 pm</td>
<td>Basics of medical imaging</td>
<td>Prof S Bunt</td>
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<td></td>
<td></td>
<td>9 am</td>
<td>Coronary Blood Flow &amp; Ischaemia</td>
<td>Dr C Bojarski</td>
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<td></td>
<td></td>
<td>4 pm</td>
<td>Body Fluid Distribution</td>
<td>Ms D Tomizzi</td>
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<td>Thur Aug 28</td>
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<tr>
<td>36 / 6</td>
<td>Tue Sept 2</td>
<td>2 pm</td>
<td>Blood vessels in the upper limb</td>
<td>Prof S Bunt</td>
<td>Blood vessels of the head and neck</td>
<td>The use of enzyme kinetics to detect markers of acute myocardial infarction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 pm</td>
<td>Blood vessels in the lower limb</td>
<td>Prof S Bunt</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>9 am</td>
<td>Bone Marrow and Haematopoiesis</td>
<td>Ms D Tomizzi</td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>4 pm</td>
<td>Erythrocytes &amp; Anaemia</td>
<td>Ms D Tomizzi</td>
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<td>Thur Sept 4</td>
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<tr>
<td>Week Yr/Sem</td>
<td>Date</td>
<td>Time</td>
<td>Lecture</td>
<td>Lecturer</td>
<td>Anatomy Lab (Wed)</td>
<td>Physiology Lab (Fri)</td>
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<tr>
<td>37 / 7</td>
<td>Tue Sept 9</td>
<td>2 pm</td>
<td>Haem &amp; Haemoglobin Metabolism</td>
<td>Dr RC Tuckey</td>
<td>Blood vessels of the upper and lower limbs</td>
<td>Human blood groups</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 pm</td>
<td>Plasma Proteins I</td>
<td>Dr P Besant</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>9 am</td>
<td>Plasma Proteins II</td>
<td>Dr P Besant</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 pm</td>
<td>Platelets &amp; Haemostasis</td>
<td>Ms D Tomizzi</td>
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</tr>
<tr>
<td>38</td>
<td>Thur Sept 11</td>
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<tr>
<td></td>
<td><strong>MID SEMESTER BREAK:</strong></td>
<td>September 15 - September 19, 2008</td>
<td></td>
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</tr>
<tr>
<td>39 / 8</td>
<td>Tue Sept 23</td>
<td>2 pm</td>
<td>Shock</td>
<td>Dr A Celenza</td>
<td>Revision and test</td>
<td>Red blood cell measurements &amp; haemostasis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 pm</td>
<td>Cardiac Failure</td>
<td>Prof J Hung</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>9 am</td>
<td>Valvular Heart Disease</td>
<td>Prof S Bunt</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>4 pm</td>
<td>Breathing (Respiratory System)</td>
<td>Prof S Bunt</td>
<td></td>
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</tr>
<tr>
<td>40 / 9</td>
<td>Tue Sept 30</td>
<td>2 pm</td>
<td>Peripheral vascular disease (last CVS lecture)</td>
<td>Prof P Norman</td>
<td>The anatomy of breathing</td>
<td>Spirometry</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 pm</td>
<td>Respiration (Ventilation I)</td>
<td>Prof H. Mitchell</td>
<td></td>
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<td></td>
<td></td>
<td>9 am</td>
<td>Physical factors affecting Ventilation</td>
<td>Dr R Fox</td>
<td></td>
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<td></td>
<td></td>
<td>4 pm</td>
<td>Respiration (Ventilation II)</td>
<td>Prof H Mitchell</td>
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<tr>
<td>41 / 10</td>
<td>Tue Oct 7</td>
<td>2 pm</td>
<td>Gases in air and Blood</td>
<td>Dr R Fox</td>
<td>Histology of the respiratory system</td>
<td>No Lab (Afternoon Memorial Service)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 pm</td>
<td>Cells and Tissues of Respiratory tract Surface</td>
<td>Dr R Fox</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>9 am</td>
<td>Diffusion of gases in Lung</td>
<td>Dr R Fox</td>
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<tr>
<td></td>
<td></td>
<td>4 pm</td>
<td>Anatomy and lung</td>
<td>Dr G Meyer</td>
<td></td>
<td></td>
</tr>
<tr>
<td>42 / 11</td>
<td>Tue Oct 14</td>
<td>2 pm</td>
<td>Lower Respiratory Tract</td>
<td>Prof S Bunt</td>
<td>The lungs and larynx</td>
<td>Respiratory and metabolic acidosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 pm</td>
<td>Upper Respiratory Tract</td>
<td>Prof S Bunt</td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>9 am</td>
<td>Regulation of O₂ Uptake</td>
<td>Prof H Mitchell</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>4 pm</td>
<td>Regulation of CO₂ and pH</td>
<td>Prof H Mitchell</td>
<td></td>
<td></td>
</tr>
<tr>
<td>43 / 12</td>
<td>Tue Oct 21</td>
<td>2 pm</td>
<td>Respiration and Disease</td>
<td>Prof H Mitchell</td>
<td>The upper respiratory tract</td>
<td>Case Study (Physiology)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 pm</td>
<td>Control of Ventilation I</td>
<td>Prof H Mitchell</td>
<td></td>
<td>Sleep Clinic (QEII)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>9 am</td>
<td>Control of Ventilation in Sleep</td>
<td>Dr D Hillman</td>
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<tr>
<td></td>
<td></td>
<td>4 pm</td>
<td>O₂ / CO₂ carriage I</td>
<td>Dr R Tuckey</td>
<td></td>
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</tr>
<tr>
<td>44 / 13</td>
<td>Tue Oct 28</td>
<td>2 pm</td>
<td>O₂ / CO₂ carriage II</td>
<td>Dr R Tuckey</td>
<td>Revision Lab</td>
<td>Case Study (Physiology)</td>
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<tr>
<td></td>
<td></td>
<td>3 pm</td>
<td>Ventilation-Perfusion Matching</td>
<td>Prof H Mitchell</td>
<td></td>
<td>Sleep Clinic (QEII)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>9 am</td>
<td>Hypoxemia</td>
<td>Prof H Mitchell</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>4 pm</td>
<td>Questions &amp; Answer Session</td>
<td>Prof H Mitchell</td>
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<td>45</td>
<td>Thur Oct 30</td>
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<tr>
<td></td>
<td><strong>STUDY BREAK:</strong></td>
<td>3rd November - 7th November</td>
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<tr>
<td></td>
<td><strong>EXAMS:</strong></td>
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<td></td>
<td></td>
<td></td>
<td>Exact date and times of the exams are set centrally and will be announced as soon as these become available</td>
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CARDIOVASCULAR AND RESPIRATORY LABORATORIES
SEMESTER 2

INDEX

Wednesday 10 am – 11.45 am Anatomy & Human Biology Dissecting Room (AM)
12 pm – 1.45 pm Anatomy & Human Biology Dissecting Room (PM)
In the Histology Lab weeks 34 & 41

Friday 10 am – 11.45 am Physiology Ground Floor Labs (AM)
2 pm – 3.45 pm Physiology Ground Floor Labs (PM)

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<td>23</td>
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<td>B*</td>
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<td>Aug 1</td>
<td>No Lab</td>
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<td>32</td>
<td>Wed</td>
<td>Aug 6</td>
<td>Mediastinum</td>
<td>25</td>
<td>A</td>
<td>B</td>
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<td>32</td>
<td>Fri</td>
<td>Aug 8</td>
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<td>A</td>
<td>B</td>
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<tr>
<td>33</td>
<td>Wed</td>
<td>Aug 13</td>
<td>The heart</td>
<td>47</td>
<td>A</td>
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<tr>
<td>33</td>
<td>Fri</td>
<td>Aug 15</td>
<td>ECG, Pulse Waves and Heart Sounds</td>
<td>57</td>
<td>A</td>
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<td>34</td>
<td>Wed</td>
<td>Aug 20</td>
<td>Histology of the cardiovascular system</td>
<td>71</td>
<td>A</td>
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<td>34</td>
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<td>Aug 22</td>
<td>Cardiac function</td>
<td>79</td>
<td>A</td>
<td>B</td>
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<td>35</td>
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<td>Aug 27</td>
<td>Blood vessels of the abdomen and pelvis</td>
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<td>A</td>
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<td>Fri</td>
<td>Aug 29</td>
<td>CVS changes in exercise</td>
<td>101</td>
<td>A</td>
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</tr>
<tr>
<td>36</td>
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<td>Sept 3</td>
<td>Blood vessels of the head and neck</td>
<td>111</td>
<td>A</td>
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<tr>
<td>36</td>
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<td>Sept 5</td>
<td>The use of enzyme kinetics to detect markers of acute myocardial infarction</td>
<td>123</td>
<td>A</td>
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<tr>
<td>37</td>
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<td>Sept 10</td>
<td>Blood vessels of the upper and lower limbs</td>
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<tr>
<td>37</td>
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<td>Sept 12</td>
<td>Human blood groups</td>
<td>141</td>
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MID SEMESTER BREAK - September 15 - September 19, 2008

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<tr>
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<th>Day</th>
<th>Date</th>
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<td>Sept 24</td>
<td>Revision and test</td>
<td>149</td>
<td>A</td>
<td>B</td>
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<tr>
<td>39</td>
<td>Fri</td>
<td>Sept 26</td>
<td>Red blood cell measurements &amp; haemostasis</td>
<td>151</td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td>40</td>
<td>Wed</td>
<td>Oct 1</td>
<td>The anatomy of breathing</td>
<td>161</td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td>40</td>
<td>Fri</td>
<td>Oct 3</td>
<td>Spirometry</td>
<td>171</td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td>41</td>
<td>Wed</td>
<td>Oct 8</td>
<td>Histology of the respiratory system</td>
<td>179</td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td>41</td>
<td>Fri</td>
<td>Oct 10</td>
<td>No Lab (Afternoon Memorial Service)</td>
<td></td>
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</tr>
<tr>
<td>42</td>
<td>Wed</td>
<td>Oct 15</td>
<td>The lungs</td>
<td>187</td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td>42</td>
<td>Fri</td>
<td>Oct 17</td>
<td>Respiratory and metabolic acidosis</td>
<td>199</td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td>43</td>
<td>Wed</td>
<td>Oct 22</td>
<td>The upper respiratory tract</td>
<td>205</td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td>43</td>
<td>Fri</td>
<td>Oct 24</td>
<td>Case Study I (Physiol)</td>
<td>215</td>
<td>A1*</td>
<td>B1*</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Sleep Clinic (QEII)</td>
<td>219</td>
<td>A2*</td>
<td>B2*</td>
</tr>
<tr>
<td>44</td>
<td>Wed</td>
<td>Oct 29</td>
<td>Revision Lab</td>
<td></td>
<td></td>
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<tr>
<td>44</td>
<td>Fri</td>
<td>Oct 31</td>
<td>Case Study I (Physiol)</td>
<td>215</td>
<td>A2</td>
<td>B2</td>
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<td></td>
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<td>Sleep Clinic (QEII)</td>
<td>219</td>
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<td>B1</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Case Study II (Online)</td>
<td>223</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Groupings used from Foundations of Clinical Practice as below:

IMED1100 Group
A1  7 – 9  Group A = A1 + A2
A2  10 – 12
B1  1 – 3  Group B = B1 + B2
B2  4 – 6
RECOMMENDED TEXTS

Physiology


Anatomy & Human Biology

Moore *et al.*, *Clinically Oriented Anatomy*, Concise Edition Preferred

OR

Drake, Vogl, and Mitchell, *Gray's Anatomy for Students* Churchill Livingstone

(A photographic atlas and colouring book may be useful for the laboratories. A list of alternative titles will be provided)
## Theme 1 Scientific Basis of Medicine (SBM)

<table>
<thead>
<tr>
<th>Strand</th>
<th>Year 1</th>
<th>IMED1100</th>
<th>Graduate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. The scientific and evidence base of</td>
<td>1. Explain scientific processes as applied to scientific and medical</td>
<td>1. Explain how physiological parameters are scientifically measured. How</td>
<td>1. Apply the scientific and evidence based approach to medicine and</td>
</tr>
<tr>
<td>medicine</td>
<td>knowledge</td>
<td>modern imaging techniques help our understanding of anatomy. To</td>
<td>practice.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>understand normal variability and accuracy of measurement.</td>
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</tr>
<tr>
<td>2. Normal structure and function</td>
<td>2. Describe aspects of normal human structure function and development</td>
<td>2. Describe the normal development, structure and function of the human</td>
<td>2. Demonstrate an in depth and comprehensive knowledge of normal human</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CVS and Respiratory system</td>
<td>development, structure, function and behaviour</td>
</tr>
<tr>
<td>3. Disordered structure and function</td>
<td>3.1.1 Describe some normal compensatory mechanisms where these illustrate the normal function of the CVS or respiratory system</td>
<td>3.1.1 Describe some normal compensatory mechanisms where these illustrate the normal function of the CVS or respiratory system</td>
<td>3. Evaluate and discuss disordered structure, function and behaviour of the human in response to internal and external factors</td>
</tr>
<tr>
<td></td>
<td>3.1.2 Discuss some disordered processes in selected diseases</td>
<td>3.1.2 Discuss some disordered processes in selected diseases where these better illustrate the importance of the normal function of the CVS or respiratory system</td>
<td></td>
</tr>
<tr>
<td>4. Pathological and clinical features</td>
<td>4. Describe basic clinical features of selected diseases</td>
<td>4. Only where it helps to illustrate and put in context the importance of understanding normal development structure and function in order to understand pathologies when these are not present.</td>
<td>4. Apply knowledge of pathological and clinical features of disease</td>
</tr>
<tr>
<td>5. Therapies</td>
<td>5. Explain basic knowledge of some therapies</td>
<td>5. Only touched on where relevant to understanding their effects on normal function</td>
<td>5. Apply knowledge of therapies to health, illness and disease</td>
</tr>
</tbody>
</table>

Draft Only July 7 - All checked except anatomy labs
AIMS AND OBJECTIVES

In general terms:
1. You are required to understand the processes, concepts and principles of the content covered in laboratories and lectures.
2. You need to know and understand these at the level of detail at which they are presented, unless specifically told otherwise. Lecture notes are guides only and may not cover the detail you are expected to know.
3. You will need to read material other than the lecture and laboratory notes in order to accomplish 1 and 2 above. YOU ARE EXPECTED TO READ OUTSIDE OF THE PRESCRIBED MATERIAL.
4. If you are required to know material that is not mentioned or covered in lectures or laboratories, this will be specifically stated.

In more detailed terms, at the end of Normal Systems students should have achieved the desired outcomes (knowledge, skills and attitudes) listed below.

**Knowledge**
1. The nature and composition of human body fluid compartments.
2. The nature, mechanisms of action and importance of neural and hormonal control systems in homeostasis.
3. The functional and anatomical organization of the autonomic nervous system and the relationship to autonomic reflexes and physiological control systems in general.
4. The physiology and anatomy of the cardiovascular system, including:
   - the histological structure of the arteries, lymphatics and veins, cardiac muscle, three layers of the blood vessel wall, and differences between muscular and elastic arteries,
   - the cardiac cycle and the heart as a pump
   - internal structure of the heart, including the layers of the heart wall, the conducting system of the heart, the valves and valve sounds
   - the blood supply of the heart and its relation to heart attack
   - the electrical activity of the heart, the normal ECG, myocardial oxygen consumption and supply via the coronary circulation.
   - the dynamics of the peripheral circulation, determinants of arterial blood pressure, control of cardiac output and the regulation of arterial blood pressure.
   - the main arteries and veins and the role of anatamoses around joints
   - the microcirculation, capillary function
   - venous and lymphatic systems, the portal venous system
   - blood flow through special regions: cerebral, limb and pulmonary circulations
   - regulation of regional blood flow and integrated responses of the circulation.
- some understanding of the features and causes of abnormal cardiac performance: arrhythmias and cardiac failure.

5. The physiology and anatomy of the pulmonary system and its relation to the cardiovascular system, including:
   - development of the lung and diaphragm
   - the anatomy of breathing
   - respiratory and olfactory epithelium and the cells that comprise these epithelia
   - the histological components of nasal conchae and the structural and functional elements of the nasal cavity
   - the anatomy and function of the sinuses and larynx
   - identification of the trachea, extra-pulmonary and pulmonary bronchi, lung lobes and lobule, alveolar ducts, terminal and respiratory bronchioles
   - identification of the cells comprising the alveolar wall of the lung and know their specific functions.
   - distinguish between a pulmonary artery and vein
   - structure of the lungs and pulmonary circulation
   - surface markings of the lungs and pleura
   - pulmonary ventilation and the mechanics of breathing
   - gaseous exchange and transport in the blood
   - relationship between respiration and acid-base status
   - features and regulation of pulmonary blood flow, ventilation/perfusion relationships
   - control of respiration
   - consequences for respiratory function of common obstructive and restrictive lung diseases
   - physiological basis of tests of lung function
   - interpretation and identification of abnormal patterns in lung function tests

Skills
Ability to recall normal ranges for important physiological variables and to recognize values that fall outside this normal range.

1. Ability to measure blood pressure by the auscultatory and palpatory methods.

2. Some ability to carry out and interpret simple blood group tests, and to recognize the major classes of blood cells.

3. Some ability to interpret abnormal ECG patterns.

4. Some ability to recognize results of lung function tests associated with restrictive, obstructive and mixed pathological conditions.
5. To become familiar with a range of modern biochemical techniques that are used in the diagnosis and treatment of disease.

6. An ability to visualize the anatomy underlying surface examination.

7. An understanding of the embryological origins of anatomical structures and where this can go wrong.

8. A basic understanding of the mechanisms and equipment available to visualise anatomy (from x-rays and MRIs etc. to laryngoscopes and endoscopes).

9. Recognition of the variability of human structure, physiology and biochemistry.

**General Attitudes**

1. To become competent at accurately and comprehensively recording what you have done, what you are doing and what you will do, why you did all these things and any observations you make during the experiment.

2. A commitment to the interpretation and evaluation of phenomena through the application of logic and the principles of physical science.

3. Some appreciation of the processes and value of practical experimentation and measurement.

4. A belief that a sound understanding of basic science underpins a full understanding of the basis of disease and provides the best hope for its diagnosis and treatment.

5. A realization of the pace of change in all areas of experimental science and the impact of new knowledge and techniques in the diagnosis and treatment of disease.

6. A desire to keep abreast of developments in research.
PREPARATION FOR ALL LABORATORY AND TUTORIAL CLASSES

_You will be expected to come to laboratory class familiar with the aims of the work to be done and with the methods and apparatus to be used._

Name badges are to be worn to all laboratory classes

The information you need is available in these Notes and in your text book. The Introduction to each class specifies what preparation you should do for tutorials.

To gain the maximum benefit from each laboratory session it is essential you come prepared and have some understanding of the areas to be covered. Before coming to each Anatomy or Physiology laboratory students must ensure the following:

1. To have read the relevant lab manual section before coming to the lab. Reprise the relevant lectures in both anatomy and physiology which, subject to timetabling and lecturer availability, should in most cases precede the relevant laboratory session.

2. Be familiar with the terms mentioned in the lab manual. This will require the use of an anatomy or physiology text book and a medical dictionary, information can also be obtained online from sites such as Wikipedia although these sites do not necessarily have the accuracy of a peer reviewed text book. If you are really stuck post a question on WebCT.

3. Have an idea of the anatomical structures mentioned in the lab book prior to coming to the lab. This will require the use of an anatomy text book or atlas. Make notes in your lab book which may include diagrams, these will be useful for revision. As the semester progresses your skills at drawing anatomical sketches should be practiced as this is a very useful ability for doctors to possess.

4. Be able to relate the physiology principals learnt to the anatomy being taught in the anatomy lab, and similarly, be able to relate the anatomical principals learnt to the physiology being taught in the physiology lab.

5. Be prepared to answer questions asked by the tutors, the labs are not didactic teaching sessions but rather interactive tutorials. Active learning, where you participate, will be retained much longer than rote learning, it also ensures you understand as well as memorize.

6. To help you in this task, at the end of each Anatomy lab you will be presented with a quiz which will have some questions on the next week’s lab to make you think and prepare. This Quiz will also contain some questions on the completed lab to cause you to reflect on what you have learned and assess whether you have really absorbed and understood the information required. Physiology labs have worksheets to be handed in for assessment a few days after the lab.
LABORATORY COATS

Laboratory Coats are compulsory for all labs for safety reasons when handling blood or other human material.

Students must supply their own lab coats, and these are available for purchase from the Guild or any workplace equipment store.

A limited number of coats are available for Hire from the front office in Physiology for $5, with a $10 deposit. Anatomy also has a few coats for hire in exceptional circumstances.

Students without lab coats will not be allowed to participate in the lab, will be marked absent, and will receive a zero grade for that class.

If students refuse to hire lab coats then the above will apply.

CONDUCT IN LABORATORY CLASSES

You must observe the following rules:

At the conclusion of a laboratory session students must remove any waste, liquid or solid from their set up and a technician must inspect your work area to ensure that it is left in a satisfactory state i.e. clean and tidy before they leave.

No food or drink to be consumed in the laboratory

Rules of conduct, based on safety requirements and on expectations of simple courtesy to fellow students and staff are displayed in the laboratory

Protective footwear must be worn in the laboratories. Definitely no thongs

Animal waste to be placed in the specially marked bins provided

All biological materials should be treated as potentially infectious/hazardous and given all due respect, especially blood

Laboratory coats should be worn, especially when dealing with blood and body fluids

Any spillages are to be reported and wiped up immediately. Spillages of biological material (i.e. blood) must be wiped up using the disinfectant solution provided. After haematology laboratories, the benches must be wiped down before leaving

Any breakages must be reported to your demonstrator and/or a technician so that it may be properly cleaned up and a replacement provided
CARDIOVASCULAR AND RESPIRATORY MODULES (PHYSIOLOGY)

LABORATORY EQUIPMENT

Calculator. It is strongly suggested that you have a simple pocket calculator available for use in the laboratory as well as with writing reports.

LABORATORY WORKBOOK

You will need a workbook of A4 format - other formats will not be accepted. A hard bound Science Exercise Book, with graph paper rules pages interleaved, is very suitable. If you choose to use an A4 format book without graph paper ruling, you will have to use sheets of loose leaf graph paper with your reports, stapled or glued into the book.

CARDIOVASCULAR AND RESPIRATORY MODULES (ANATOMY)

Please leave all bags on the shelves provided inside the dissecting room doors then move promptly to your assigned table.

Four tables will be laid out on each side of the dissecting room. Eight tables in total. A timer will beep at 20 minute intervals indicating that you should move to the next table.

One table in each set will be based on clinical correlates of anatomy and may include radiology and imaging methods and self testing material.

Please make sure you wear gloves (provided) to protect yourself against the minimal chance of catching Creuzfeldt Jacob disease from the cadavers.

Wash up and remove your white coat before leaving the dissecting room.
DISSECTION ROOM (DR) RULES

1. An Anatomy Licence must be obtained before entry to the Dissection Room.

2. In the Anatomy labs you must wear a clean, graffiti free, white coat, a name badge and closed shoes.
   Coats can only be hired in exceptional circumstances and availability cannot be guaranteed.
   Repeated failure to wear appropriate clothing may result in exclusion from the laboratory.

3. Protective footwear must be worn, i.e. - no thongs or sandals.

4. All cadavers and wet specimens must be covered with a wet cloth and a plastic cover when unattended.

5. Used scalpel blades must be placed in the sharps containers provided.

6. Please consult a technician before moving any bottles, skeletons etc - they can be very heavy and easily damaged.

7. Wet prossected specimens are extremely valuable and take many weeks to prepare. They must not be further dissected as this may render them useless to other classes.

8. The skeletons are very delicate, please handle with care and do not disarticulate them.

9. No wet specimens or bottled specimens are to be taken from the Dissection Room. Any attempt to do so may lead to instant dismissal from the course.

10. The Dissection Room and adjoining washroom must be left in a clean and tidy condition. All paper towels and other rubbish must be placed in the bins provided.

11. We are dependent on body donations for our teaching, any inappropriate behaviour in the dissecting room will result in exclusion from the laboratory. In severe cases this can result in expulsion from the course.

12. What occurs in the DR must be treated with sensitivity and confidence maintained. Be aware of the effect your descriptions might have outside of the teaching context.

13. No photography allowed (including mobile phone cameras)

For any assistance, please ask one of the Dissecting Room technical staff.
Follow the instructions of the semester co-ordinator and the Dissecting Room technical staff at all times - if in doubt - ASK.
SAFETY PROCEDURES
FOR HANDLING POTENTIALLY INFECTIOUS MATERIALS
e.g. Human blood and urine

ALL BLOOD and urine specimens should be treated as infectious.

When handling such products students should:

• Wear buttoned up laboratory coats. Students without lab coats will be excluded from the class and considered absent.
• Wear footwear that covers the foot (no thongs or sandals).
• Have hair tied back from the face.
• Wear disposable gloves (provided by the Physiology Department)
• Wear protective glasses (provided by the Physiology Department)
• Inform demonstrator of the presence of any broken skin on the hands (this should be covered by a bandaid and protective gloves; if extensive the student should not participate)
• Not touch eyes, mouth or face
• Not eat or drink in the laboratory
• Not do anything that would cause blood to splash or aerosolise
• Dispose of blood or contaminated objects promptly into appropriate containers
• Dispose of sharps promptly into appropriate sharps containers
• Report immediately to demonstrator any accident involving blood, e.g. spillage, contamination of skin, eyes or mouth

As some of your laboratory classes will involve the collection of student blood and urine samples, any students knowing themselves to be carriers of blood borne infection should contact the Faculty Infection Control Officer, Dr Liam O'Connor (Phone: 9346 2496) as soon as possible to discuss the risks involved.

FIRE

The continuous sounding for the FIRE ALARM or evacuation HORN defines the precise moment when emergency evacuation should commence. All students should at that time make their way to the nearest exit in a quiet, orderly fashion and thereafter assemble on the grassed area immediately in front of the building so that it may be ascertained that all students have in fact left the building. Please familiarise yourself with the location of exists to the building.
Plagiarism is the copying of material from another person without acknowledgment, and it includes copying from textbooks and from laboratory reports from your current colleagues and students from previous years. Copying of material from other workers without acknowledgment cannot be condoned in any scientific community, and if detected could have very serious consequences as far as your academic record is concerned and may wreck your future prospects in research. It is, of course, a form of cheating and insofar as the marks for your laboratory reports are included in your final assessment, plagiarism from the work of others, including other students, in the preparation of these reports is as serious as cheating in the actual written exam.

The Macquarie Dictionary defines plagiarism as “the appropriation or imitation of another’s ideas and manner of expressing them, as in art, literature, etc., to be passed off as one’s own.” “Passed off as one’s own” is the important phrase. It is quite legitimate to quote from some other clearly recognised source as support of your arguments, if the source is readily available to others, but the quote MUST include an unambiguous reference to the source and an indication that it is NOT your own work. In science, of course, almost every statement is based on another person’s ideas, and most of what you write will be paraphrasing information from someone else. We expect that you will synthesise your reports from material from a variety of sources, textbooks, lecture notes, discussions at tutorials, discussions amongst yourselves, possibly even research papers. We do not expect you to attribute everything you write to the original or even the proximate source, especially for well established facts and theories. However, what you write must be in your own words, and if you copy word for word a paragraph from a research paper, textbook, or even a handout, you must attribute the quotation to the source. Any quotations must follow the original material exactly, with clear indications where the quotation starts and finish and should generally be short. We would not necessarily consider extensive quotation as plagiarism if the source was given, but neither would we give any added marks and we would certainly take off marks if the included material was not completely relevant.

We do not suggest that you must always work alone on a report. You work in groups in the laboratory and there are many advantages in working together as a group in analysing the results and the problems. Obviously the members of such a group will present similar reports with similar results and conclusions. But each report must be wholly prepared by each individual and must be presented in that student’s own words.
and where the data are presented graphically, every graph and diagram must be the students own work. Reports which include photocopies of the data analysis will automatically receive zero marks.

If plagiarism is detected, the following penalties will apply:

1) If a number of students hand in identical reports, one of these will be assessed and each student will get a mark calculated by dividing this assessment by the number of students handing in the identical reports. The students involved will be interviewed by the course controller and may be excluded from the course if the offence is repeated. The assumption here is that all students colluded in the preparation of the report. Even if it can be shown that a single student prepared the report and allowed the others to copy it, all students involved will be treated equally.

2) If identical reports are handed in and it is proven that one report was copied without the permission of the original author, the copied report will be given zero marks and the student involved will be reported to the faculty for disciplinary action. This may include exclusion from the course.

3) If reports from a previous year are copied, or if material from a text or other source is repeatedly presented without acknowledgment the student will receive zero marks. The reason for this unsatisfactory performance will be reported to faculty

EACH REPORT MUST CONTAIN A STATEMENT SIGNED BY THE STUDENT THAT THE MATERIAL IS ALL HIS/HER OWN WORK. IN THE ABSENCE OF THIS STATEMENT THE REPORT MAY NOT BE MARKED.
CARDIOVASCULAR MODULE
Anatomy Laboratory

Wednesday, Week 1

Introduction to the Laboratory and the course
CARDIOVASCULAR MODULE
Anatomy Laboratory

Wednesday, Week 2

Mediastinum
Material used in this Laboratory:

Two sets of four tables

Station 1 Skeleton, prosections of intercostal muscles, ribs chest X rays

Station 2 Lateral mediastinum, prosections

Station 3 Deep dissections of the neck, thorax showing aortic arch

Station 4 Posterior chest wall showing oesophagus, sympathetic chain etc.
Laboratory class: The chest wall and mediastinum

Read and study these notes and use your textbook to become familiar with the contents BEFORE you come to the lab.

The laboratory will be divided into four sections, students will rotate around the four stations (one on each side of the laboratory) when the beeper goes off. Please stay in your groups to avoid too many students at any one station.

If there is time, in the last 20 minutes of the lab form into groups around each demonstrator and participate in a discussion with your colleagues and tutor about the mediastinum, its parts and the contents of each part.

Be prepared to demonstrate the following structures to the group, both on a prosection and the surface marking where relevant.

- Sternum, manubrium, body of sternum, xiphoid process, manubriosternal joint (sternal angle), xiphisternal joint, right brachiocephalic vein, superior vena cava, azygos vein, inferior vena cava, trachea, oesophagus, root/hilum of the lung, bronchus, pulmonary artery, phrenic nerve, vagus nerve, intercostal veins, arteries and nerves, sympathetic trunk, right atrium of the heart, arch of the aorta, left subclavian artery, left common carotid artery, sympathetic nerves to the heart, recurrent laryngeal nerve, ligamentum arteriosum, hemiazygos vein, Left ventricle of the heart, Internal jugular vein and subclavian vein, brachiocephalic vein, thoracic duct, descending aorta, oesophageal arteries, bronchial arteries, internal thoracic artery, fibrous pericardium.

Outcomes

1. The student will be able to define the mediastinum and its boundaries and subdivisions in relation to the skeleton of the thorax.
2. The student will be able to identify structures that can be seen on the left and right aspects of the mediastinum.
3. The student will be able to describe the layers that comprise the superior mediastinum.
4. The student will be able to outline the shape, boundaries and contents of the posterior mediastinum.
5. The student will be able to identify the components of the autonomic nervous system visible in the thoracic cavity, in particular the vagal nerves and the sympathetic chain.
Station 1 MEDIASTINUM GENERALLY

Examine the skeletons and prosections that show the anterior chest wall:

Parts of the sternum: Manubrium, body and xiphoid process. Which costal cartilages join and what are the vertebral levels of:

<table>
<thead>
<tr>
<th>Costal cartilage</th>
<th>Vertebral level</th>
</tr>
</thead>
<tbody>
<tr>
<td>The top of the manubrium</td>
<td></td>
</tr>
<tr>
<td>Manubriosternal joint (sternal angle)</td>
<td></td>
</tr>
<tr>
<td>Xiphisternal joint</td>
<td></td>
</tr>
</tbody>
</table>

Correlate the above with the appearance of a lateral X-ray of the chest and note the domes of the diaphragm and the narrow space behind the diaphragm. At what vertebral and sternal level is the base of the heart where it rests on the central portion of the diaphragm?

Examine the size and shape of the mediastinum in prosections and chest X-rays. Note the variation in size and position of the mediastinum. Also be aware that it lies behind the sternum, and that it extends a little to the right of the sternum all the way down. On the left hand side the left ventricle of the heart is responsible for it extending much further to the left in its lower part. Draw the outline of the mediastinum and its components (Superior, middle and posterior) on the anterior and lateral outlines of the thorax provided.
Station 2 THE LEFT AND RIGHT SIDES and MIDDLE MEDIASTINUM
Examine the left and right aspects of the intact mediastinum. Some prosections will have the pericardium opened and others will not.

THE RIGHT SIDE OF THE MEDIASTINUM
On the right hand side note that the veins predominate: Right brachiocephalic, superior vena cava (SVC), azygos vein, inferior vena cava (IVC). They all drain to the right atrium of the heart. Also note that the trachea and the oesophagus are easily seen. The root/hilum of the lung is cut through and you can identify the bronchus (by its cartilage rings), the pulmonary artery and veins. The phrenic nerve is seen running directly down along the side of the mediastinum to the diaphragm. The vagus nerve is not so readily seen, but in the upper part it passes behind the phrenic nerve and heads for the hilum of the lung. Posteriorly, the intercostal veins, arteries and nerves can be seen in each space and the sympathetic trunk runs all the way down on the sides of the vertebrae.

Draw a diagram of the right hand side of the mediastinum and label the major structures listed:
R Brachiocephalic v
SVC
Azygos vein
Intercostal veins
Phrenic nerve
Trachea
Hilum of lung
Vagus nerve
Oesophagus
Right atrium of the heart
IVC
THE LEFT SIDE OF THE MEDIASTINUM

On the left hand side note that the arteries predominate. The arch of the aorta is obvious, with its left subclavian and left common carotid branches. The descending aorta is usually apparent throughout. The left side of the heart bulges markedly to the left. The components in the hilum of the left lung can be identified. The aortic arch is crossed by the phrenic and vagus nerves and, on some prosections, sympathetic nerves descending from the neck can be seen crossing the aortic arch. Look closely at the vagus nerve crossing the arch on its way to the hilum of the lung. It gives off a large branch (the recurrent laryngeal nerve) which hooks under the arch of the aorta behind the ligamentum arteriosum. Posteriorly, the intercostal veins, arteries and nerves, and the sympathetic trunk can be seen as on the right side. On some prosections, the mediastinum can be pushed away to the right and the intercostal veins can be seen joining the hemiazygos vein.

Draw a diagram of the left hand side and label the major structures listed:

Aortic arch
Left subclavian artery
Left common carotid
Vagus nerve
Phrenic nerve
Sympathetic branches
Intercostal veins
Hilum of lung
Left ventricle of the heart
Oesophagus

MIDDLE MEDIASTINUM

The middle mediastinum contains the heart (which will be studied in detail next week). However, today you should note that the fibrous pericardium is firmly attached to the central portion of the diaphragm. What is the significance of this for the position of the heart during the respiratory cycle?
Station 3 SUPERIOR MEDIASTINUM AND AUTONOMICS

What structures define the lower boundary of the superior mediastinum?

Superior/middle mediastinum

Superior/posterior mediastinum

On a prosection where the manubrium has been removed observe the following structures in the superior mediastinum:

A) The Internal jugular and subclavian veins unite to form the brachiocephalic veins (this happens behind the medial ends of the clavicles). The left and right brachiocephalic veins unite to form the SVC on the right side, and the SVC descends a short distance to the right atrium of the heart.

List the veins from the neck and thorax, which join the brachiocephalic veins:

B) Arch of the aorta and its 3 branches:

Examine the aortic angiograms that show the aorta and its main branches.

Confirm the relations of the vagus and phrenic nerves to the plane of the veins and arteries.

C) Behind the aortic arch is a group of 4 vertical structures

The trachea and oesophagus are large and easily seen in deeper prosections. The left recurrent laryngeal nerve can be followed from where it leaves the vagus nerve on the arch of the aorta.

Where is the right recurrent laryngeal nerve?

The 4th member of the group of 4 (the thoracic duct) is not so easily seen in the superior mediastinum, but you will be able to see it in the posterior mediastinum.
In preparation for your next physiology lab you should note the position of the sympathetic chain and the vagal nerve carrying parasympathetic output to the viscera.

What is the distribution of the phrenic nerve?
Motor

Sensory

What does the vagus nerve do for the:
Heart?

Lungs?

Oesophagus?

GIT in the abdomen?
Station 4 POSTERIOR MEDIASTINUM

Study prosections where the heart has been removed or is very mobile, and look at the structures that descend behind the heart.

From right to left: the azygos vein, the thoracic duct and the descending aorta lie in front of the thoracic vertebral bodies. The oesophagus lies in front of these structures and is directly behind the left atrium of the heart. Note that there is a plexus of nerves running on the oesophagus. Trace those nerves into continuity with the vagus nerve at the hilum of the lung.

Carefully attempt to follow the thoracic duct into the superior mediastinum where it crosses to the left hand side.

On the deepest prosections examine the branches of the descending aorta:
- Posterior intercostal
- Oesophageal
- Bronchial

Also on the deepest prosections, examine the structures that are associated with the thoracic wall (the first three below run horizontally in the intercostal spaces):
1. The venous drainage of the chest wall. The posterior intercostal veins joining the azygos vein on the right, and the hemiazygos on the left. The hemiazygos veins cross the midline to empty into the azygos vein.
2. The arterial supply of the chest wall. The posterior intercostal arteries branching from the aorta
3. The nerve supply of the chest wall. The ventral rami of thoracic spinal nerves
4. The sympathetic trunk runs vertically beside the vertebral bodies and gives medial branches to viscera and lateral branches to the body wall.

Where do the anterior intercostal arteries arise and how do the anterior intercostal veins drain? Look at the posterior aspect of the anterior chest wall and also the angiograms on display.

How does the internal thoracic artery end?
CARDIOVASCULAR MODULE
Physiology Laboratory

Friday, Week 2

SYMBIOSIS: Autonomic nervous system
The autonomic nervous system and the regulation of cardiovascular function

BACKGROUND

The circulation of blood around the body depends ultimately on the output of the heart and on the integration of the flow of blood into tissues, which is regulated in accordance with the needs of the tissue whether for gas exchange, for substrate delivery or for some other physiological process. This concept is encapsulated in the two relationships:

\[ CO = HR \times SV \]

(where CO is cardiac output, HR is heart rate, and SV is stroke volume)

\[ BP \propto CO \times SVR \]

(BP is mean arterial blood pressure and SVR is systemic vascular resistance or peripheral resistance)

The role of the Autonomic Nervous System (ANS)

All parameters in the above relationships are controlled by the sympathetic and parasympathetic fibres of the autonomic nervous system. The heart receives both types of innervation, such that changes in ANS activity result in changes in HR and SV. The smooth muscle of small muscular arteries, arterioles and veins receives innervation mainly from the sympathetic system, such that changes in ANS activity result in changes in SVR.

Thus in the heart both heart rate and contractility are controlled by the autonomic nervous system, which in turn means control of CO (relationship #1). The control of flow through the resistance vessels, which are responsible for peripheral resistance, obviously also affects the amount of blood flowing into the capillaries of various tissues. Thus the resistance vessels act as “stopcocks” for the outflow of blood from the high-pressure arterial tree. Control of their resistance therefore plays a key role in the maintenance of normal arterial blood pressure (relationship #2).

Note: While the ANS is a very important regulator of cardiovascular control, it is not the only means of regulation.

In many tissues the flow varies considerably, depending on the metabolic requirements of the tissue at any moment. For example in skeletal muscle flow can vary from about 1 Litre per minute at rest, to nearly 25 Litres per minute in elite athletes during intense activity. Modulation of blood flow by mediators acting locally may be overridden by the effects of the autonomic innervation of blood vessels and the influence of circulating hormones such as adrenaline, noradrenaline and angiotensin II. These are not the only factors affecting tissue blood flow and you will learn about others later.
Objectives

This class will introduce you to a computer simulation software program that will help you to understand the physiological control of the circulatory system. SymBioSys is a large program with many features. Today you will work through an exercise that has been designed for you, to reinforce your knowledge of the roles of the sympathetic and parasympathetic innervations of the heart and blood vessels. However you might want to arrange with the laboratory technicians for additional access to work through other problems relating to the circulation. You will also use the program later this semester to work through some exercises on other aspects of cardiovascular physiology and on the physiology of respiration.

First, make sure that the program is set up on your computers and running. You will need to tabulate your results in your own laboratory notebooks. Such tables are provided on the pages at the end of this lab (within the report questions) and are also available on WebCT.

The opening screen contains three windows, as illustrated below:

Spend a few minutes inspecting the screen. Notice that there are a tutorial, exercises, test questions and a help menu in the large main window. On the lower left is the Tools window including Blood Withdrawal (Phlebotomy) and a Drug and Fluid Infusor (for injecting things). However for now, go to the upper left Curriculum window and double-click on the folder “Custom Exercises”, then on “Autonomic Control of CVS 1”. Work through the exercise in the main window following the instructions as you go.
Some important points:

Note the padlocks – unlock them if you want control of a parameter, otherwise the program has control and adjusts them as required according to the afferent inputs. That is, a locked padlock is like a functioning ANS, an unlocked padlock means you control the ANS.

Note the definitions used - e.g. SVR (systemic vascular resistance) for peripheral resistance, LVEDP (left ventricular end diastolic pressure, LVEDV (left ventricular end diastolic volume) etc

Construct tables in your own notebooks like the one below to record the information obtained.

<table>
<thead>
<tr>
<th>PSNS Tone</th>
<th>SNS Tone</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Heart Rate (b/min)</td>
<td>0.1</td>
</tr>
<tr>
<td>Cardiac Output (l/min)</td>
<td></td>
</tr>
<tr>
<td>SVR Torr/l/min</td>
<td></td>
</tr>
<tr>
<td>LVEDP (mmHg)</td>
<td></td>
</tr>
<tr>
<td>BP (s/d) (mmHg)</td>
<td></td>
</tr>
<tr>
<td>Pulse pressure (mmHg)</td>
<td></td>
</tr>
<tr>
<td>Stroke Volume (ml)</td>
<td></td>
</tr>
</tbody>
</table>

You can extract most of the information from the table in Symbiosys, but you will need to scroll up to the pressure volume loop to record the LVEDP. At the same time observe what is happening to the traces. You can enlarge the main window by dragging its left border. The scroll bar disappears if you enlarge the window but will reappear when you try to use it.

You need to calculate the pulse pressure and stroke volume from the other variables.
Pulse pressure is (systolic blood pressure minus diastolic blood pressure).
Stroke volume can be calculated several ways. The easiest is cardiac output ÷ heart rate.

EXERCISE 1. The effect of varying parasympathetic tone.
1. Unlock the padlocks and set sympathetic tone to 0.1, and parasympathetic tone to 0. Wait a minute or so for the variables to stabilise then record them in your table (when the padlocks are locked the parameter is adjusted automatically by the program; when you unlock the padlock it is under your control to set as required).
2. Keeping sympathetic tone constant at 0.1, systematically vary the parasympathetic tone to 0.25, 0.5, 0.75 and 1.0. At each step wait until the variables have stabilised before recording them.
3. When you have finished, restore the normal physiology by clicking on the Simulation menu at the top of the screen then clicking on **Reset Physiological State**. This step also locks all padlocks.

**QUESTION:** What are the main reasons for the decrease in cardiac output when parasympathetic tone increases?

**EXERCISE 2. The effects of varying sympathetic tone.**
Before you begin prepare a new table with the same variables as before.

1. Unlock the padlocks and set parasympathetic tone to 0.36, and sympathetic tone to 0.1. Wait a minute or so for the variables to stabilise then record them in your table.
2. Keeping parasympathetic tone constant at 0.36, systematically vary the sympathetic tone to 0.25, 0.5, 0.75 and 1.0. At each step wait until the variables have stabilised before recording them.
3. When you have finished each step, restore the normal physiology by clicking on the Simulation menu at the top of the screen then clicking on **Reset Physiological State**.

**QUESTION:** Does the diastolic BP increase relatively more than the systolic BP when sympathetic tone increases? What happens to SVR and why?

**QUESTION:** From the first two exercises, can you see a relationship between pulse pressure and stroke volume? Plot these parameters against each other on a graph. Can you explain the reason for any relationship you find?

**EXERCISE 3. The function of the autonomic nervous system after haemorrhage**
Reset the Physiological State, then go to the upper left Curriculum window, widen it if needed and then double-click on the folder “Custom Exercises”, then on “Autonomic Control of CVS 2”. Follow the instructions to set up a haemorrhage scenario.

Construct a new table with three data columns and with the same variables as before, but add extra rows for Pms, Sympathetic Tone and Parasympathetic Tone (Pms = Mean Systemic Pressure).

1. Record the variables before you begin (make sure that you have reset the normal physiological parameters).
2. Without releasing the padlocks (so that the normal autonomic control is intact) withdraw 1 litre of blood using the maximum rate of withdrawal. This simulates a large loss of blood, as when a major blood vessel has been severed. Observe what happens to the traces, and when things have stabilised record the variables.
3. Reset and wait for the variables to return to starting values.
4. Now release the padlocks and withdraw 1 litre of blood again. Record as before.
5. Mop up any blood you have spilled on the floor. Alert a relative.

**QUESTION:** What are the main differences in the response to haemorrhage when the ANS is functioning compared with when it is inactivated, and how can you explain the effects of the ANS in response to
haemorrhage?

EXERCISE 4. The effect of some drugs on the cardiovascular system.

Before you begin prepare a new table with the same variables as before but with 6 data columns (two for each drug) for BEFORE DRUG AND AFTER DRUG. Then you need to RESET again, and arrange to inject some drugs. Go to the “Tools” Window and click on “Drug and Fluid Infusor”. In the Infusor window click the red “+” then select Atropine from the drop-down list. Point at the new bar on the window, and use the arrow on the left to change the dose to 1 mg and the frequency to single dose. Now add Esmolol from the Pharmacy drop-down list, then change the infusion rate to 150 mg/min. Finally add Phentolamine to your Infusor window and change the dose to 5 mg in a single injection. You can shift this window around on the screen if it obscures your observations. In this exercise you will inject the drugs selected to observe the effects of blocking the sympathetic and parasympathetic inputs to the cardiovascular system. To inject a drug click on the stop sign, and to stop click the stop sign again.

Interpretation of the results can be aided if physiological feedback is blocked. Thus you will unlock the padlocks, and any changes you observe are then direct effects of the drugs rather than feedback effects of the ANS in response to the changes induced by the drugs.

1. Unlock the padlocks and set parasympathetic tone to 0.5, and sympathetic tone to 0.2. Wait a minute or so for the variables to stabilise then record them in your BEFORE column.
2. Now inject ATROPINE, then wait until the variables have stabilised before recording them.
3. Now restore the normal physiology by clicking on the Simulation menu at the top of the screen then clicking on Reset Physiological State.
4. Unlock the padlocks and set parasympathetic tone to 0.36, sympathetic tone to 0.5. Wait a minute or so for the variables to stabilise then record them in your second BEFORE column.
5. Now infuse ESMOLOL and record the effects.
6. Repeat steps 3 & 4
7. Infuse PHENTOLAMINE and record the effects.

QUESTION: From your observations what can you deduce about the effects of the drugs you have injected? Give reasons for your answer.

QUESTION: What mechanisms other than the autonomic nervous system regulate cardiac output? What happens for example in a person after a heart transplant where the innervation to the heart has been severed?
I hereby certify that I have read the plagiarism statement in the unit manual, that I have made myself familiar with the university’s policy on plagiarism and that the report/essay/assignment submitted does not contain plagiarism.

Signature .............................. Date ........................................................

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**LAB REPORT 1: Symbiosis – Autonomic Nervous System**

**EXPERIMENT 1:**

1) Complete the following Table showing clearly what happened to the monitored variables when you systematically varied parasympathetic tone.

<table>
<thead>
<tr>
<th>PSNS Tone</th>
<th>SNS Tone</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>0.1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Heart Rate (b/min)</th>
<th>Cardiac Output (l/min)</th>
<th>SVR (Torr/l/min)</th>
<th>LVEDP (mmHg)</th>
<th>BP (s/d) (mmHg)</th>
<th>Pulse pressure (mmHg)</th>
<th>Stroke Volume (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2) What are the main reasons for the decrease in cardiac output when parasympathetic tone increases?
3) Complete the following Table showing clearly what happened to the monitored variables when you systematically varied sympathetic tone.

<table>
<thead>
<tr>
<th>PSNS Tone SNS Tone</th>
<th>0.36</th>
<th>0.36</th>
<th>0.36</th>
<th>0.36</th>
<th>0.36</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.1</td>
<td>0.25</td>
<td>0.5</td>
<td>0.75</td>
<td>1.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Heart Rate (b/min)</th>
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4) Does the diastolic BP increase relatively more than the systolic BP when sympathetic tone increases?

5) From the first two exercises, can you see a relationship between pulse pressure and stroke volume? Attach a plot these parameters against each other.

6) Explain why this relationship occurs?
7) Complete the following table comparing the response to haemorrhage with and without a functioning ANS.

<table>
<thead>
<tr>
<th>Haemorrhage</th>
<th>Normal</th>
<th>ANS functional, -1L blood, 10 L/hr</th>
<th>ANS inactivated, -1L blood, 10 L/hr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Rate (b/min)</td>
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<tr>
<td>Cardiac Output (l/min)</td>
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<td>SVR (Torr/l/min)</td>
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<td>BP (s/d) (mmHg)</td>
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<td>Pulse pressure (mmHg)</td>
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<td>Stroke Volume (ml)</td>
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<td>PMS (mmHg)</td>
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<tr>
<td>SNS tone</td>
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8) Describe what happened to blood pressure in the two situations (with and without ANS).
9) Explain the differences in blood pressure homeostasis in terms of how the other variables responded with and without an ANS.

10) Table of drug effects.

<table>
<thead>
<tr>
<th>Haemorrhage</th>
<th>Normal</th>
<th>Atropine</th>
<th>Normal</th>
<th>Esmolol</th>
<th>Normal</th>
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</table>
10) From your observations after infusing drugs, what do you deduce about the action of atropine? Does it have effects as if it is a sympathomimetic or parasympathomimetic drug?

11) From your observations after infusing drugs, what do you deduce about the action of esmolol? Does it have effects as if it is a sympathomimetic or parasympathomimetic drug?

12) From your observations after infusing drugs, what do you deduce about the action of phentolamine? Does it have effects as if it is a sympathomimetic or parasympathomimetic drug?
CARDIOVASCULAR MODULE

Anatomy Laboratory

Wednesday, Week 3

The heart
Material for this laboratory

Two sets of four tables

STATION 1: Orientation & External Features of the Heart, chest X-ray, CT to show heart

STATION 2: Examination of the Interior of the Human Heart

STATION 3: Dissection of the Sheep’s Heart (section off)

STATION 4: Surface Anatomy – crayons, stethoscopes, partitions for privacy
NS 100  Cardiovascular system

Laboratory class: The Heart

Read and study these notes and use your textbook to become familiar with the contents BEFORE you come to the lab.

Outcomes

1. The student will be able to describe the position and orientation of the heart both in the thoracic cavity, and also with respect to an articulated skeleton.
2. The student will be able to describe the coronary circulation and be able to identify the origins and main branches of the coronary vessels.
3. The student will be able to dissect a sheep heart, and to recognise and explain the main features of each of the chambers, valves and major vessels.
4. The student will be able to correlate the features of the sheep heart with those of the human and realise that they are essentially the same.
5. The student will be able to mark the standard outline of the heart on the anterior chest of a fellow student, and to be able to locate the heart chambers, valves and major vessels in relation to that outline.
6. The student will be able to locate the positions on the chest wall where each heart valve can be auscultated, and to relate the timing and locations of these valve sounds to the cardiac cycle.

Station 1 ORIENTATION AND EXTERNAL FEATURES OF THE HEART

Use prosections of the thorax to examine the heart in situ

Observe the features seen on the anterior aspect and record them on the outline below:

Right atrium  SVC
IVC
Pulmonary trunk
Left ventricle
Right auricle
Right marginal artery
Anterior interventricular a
Left coronary artery
Left marginal artery
Right ventricle
Ascending aorta
Coronary sulcus
Right coronary a.
Interventricular sulcus
Left auricle
Circumflex artery
Take an isolated heart and orientate it.
The right atrium and vena cavae to the right.
The great vessels emerging superiorly.
The apex of the heart (ventricles) to the front left.
Hold the heart by forming a ring with the fingers and thumbs of both hands wrapped around the coronary sulcus. Note that on one side of that ring are the two ventricles and on the other side are the atria and great vessels.

**CORONARY VESSELS**
Follow the course and distribution of the left coronary artery (anterior interventricular, circumflex and left marginal arteries), and the right coronary artery (right marginal, posterior interventricular arteries).
Note that both arteries arise from the ascending aorta and emerge onto the anterior aspect of the heart under cover of their respective auricle. Insert your finger behind the aorta and pulmonary trunk and in front of the great veins (transverse pericardial sinus) and note the course of the coronary arteries in relation to this space.
Which arteries form anastomoses? ____________________________________________
Which arteries are end arteries? ____________________________________________
Examine the cardiac veins and draw them on the diagram provided alongside their accompanying arteries. Follow them to the coronary sinus in the posterior part of the coronary sulcus.
Great cardiac vein
Small cardiac vein
Middle cardiac vein
Oblique cardiac vein
Anterior cardiac vein
Coronary sinus
STATION 2 EXAMINATION OF THE INTERIOR OF THE HUMAN HEART

RADIOGRAPHY

Study the isolated human hearts and those seen in the prosections. Go through them chamber by chamber, where possible, and identify all the same features seen in the sheep heart.

POSITIONS OF THE VALVES

Again, place your hands into a ring around the coronary sulcus and realise that all the 4 valves you have seen lie in the plane formed by that ring (fibroelastic skeleton)

Which valve is at the top left extreme?

Which valve is next from the top?

Which valve is next?

Which valve is at the bottom right extreme?

DRAW the valve positions on the diagram below

In which directions would you expect the blood flow to carry the sounds of those valves?

- Pulmonary and aortic
- Left and right atrioventricular

What is the difference between an MRI, thin film X-ray and CAT scan? How are angiograms obtained?

Can you identify some artifacts on the films?

Examine the catheters provided; many arterial blockages are now opened by passing a catheter from the Iliac artery to the heart.
STATION 3 DISSECTION OF THE SHEEP HEART

RIGHT ATRIUM.
Orientate the sheep’s heart and identify the right atrium and vena cavae. Open the right atrium by cutting with scissors (much safer and easier than using a scalpel) from the SVC to the IVC and remove the blood clot (if present). Examine the internal aspect of this chamber and identify: musculae pectinati, crista terminalis, and the smooth walled part derived from sinus venosus. Identify the openings into the right atrium: SVC, IVC, Coronary sinus, Atrioventricular orifice. Find the fossa ovalis in the interatrial septum and imagine the blood flowing from the IVC into the foramen ovale, whilst blood from the SVC goes directly into the AV opening in the fetal circulation. The sinu-atrial node is not visible but lies near the junction of the crista terminalis and the SVC (the atrioventricular node lies in the inter-atrial septum near the AV orifice).

RIGHT VENTRICLE
This chamber can be opened either by inserting one blade of your scissors into the AV orifice and cutting down the right margin of the heart, or by cutting just below the coronary sulcus and just to the right of the interventricular groove and opening the resultant flap. Remove the clot of blood and examine the interior. Trabeculae carnae, smooth infundibulum near the pulmonary valve, papillary muscles and chordae tendineae leading the 3 cusps of the AV valve (tricuspid valve). Note that each cusp receives chordae from two papillary muscles.
What causes these cusps to open and close?

What is the function of the papillary muscles and chordae tendineae?

PULMONARY TRUNK
Look at the pulmonary valve from the ventricular aspect and insert one blade of scissors between two cusps on the anterior aspect and open the pulmonary trunk by cutting upwards. Examine the valve cusps and identify the lunule and nodule on each, and the space/sinus behind each cusp.
What causes these cusps to open and close?
LEFT ATRIUM
Open the left atrium by cutting transversely between the pulmonary veins and remove the blood clot. Note that most of the interior surface is smooth apart from area including the left auricle. Identify the interatrial septum and correlate its position with the fossa ovalis as seen in the right atrium. Try to insert a blunt probe through the upper part of the fossa ovalis to connect the two atria (this should be possible in about 20% of hearts examined)

LEFT VENTRICLE
Open the left ventricle by cutting between the cusps of the Left AV valve and down the left margin of the heart, remove the blood clot and examine inside this chamber (trabeculae carnae, papillary muscles chordae tendineae). The AV (mitral) valve has only two cusps and the aorta takes origin behind the larger posterior cusp (note the smooth walled area just inferior to the aortic valve). Why is the left ventricular wall almost 3 times as thick as the right ventricle wall?

__________________________

Is the volume of the left ventricle the same as that of the right ventricle? ____________
Should it be? ___________________________ Why? ___________________________
What causes blood to flow from the atria to the ventricles? ___________________________

AORTA
Opening the aorta is somewhat destructive because it passes behind the pulmonary trunk, but do so with an incision passing between two semilunar cusps. Open out the ascending aorta and note the same features of the aortic valve as seen on the pulmonary valve. However, note that behind the two of the cusps (left and right aortic sinuses) there are origins of coronary arteries. At what stage of the cardiac cycle does blood pass into the coronary arteries? _________________

__________________________

What force causes blood to flow in the coronary arteries?

__________________________

Do you think this is the most effective way of perfusing the heart muscle? _________________
Why? ___________________________
Examine the ligamentum arteriosum in both the pulmonary artery and the aorta. Revise your knowledge of the role of the ductus arteriosus in the fetal circulation. ___________________________

__________________________

(Remember the fetal pig!)
STATION 4 SURFACE ANATOMY – Form groups of three where one person can remove their shirt and act as the subject. You will be provided with diagrams to help you.

Palpate and mark the following bony features of the chest wall

Suprasternal notch (vertebral level ? ________)

Heads of clavicles

Sternal angle (vertebral level ? ____________)

2\textsuperscript{nd} costal cartilage

3\textsuperscript{rd} costal cartilage

4\textsuperscript{th}, 5\textsuperscript{th} and 6\textsuperscript{th} costal cartilages

Xiphoid process

Xiphisternal joint (vertebral level ? ________)

Mark the position of the 12\textsuperscript{th} thoracic vertebra (lower end of the thoracic aorta)

Outline of the heart

Mark points

A) 1cm to the right of the xiphisternal joint (IVC pierces diaphragm)

B) 1cm to the right of the sternum on the 3\textsuperscript{rd} costal cartilage

C) 1cm to the left of the sternum in the 2\textsuperscript{nd} intercostal space

D) In the mid clavicular line (~nipple line) in the 5\textsuperscript{th} intercostal space

Join the points A – B – C – D

Be aware that these are the standard markings of the heart in supine cadaver. The heart lies somewhat lower in an erect living subject, and the moves with the diaphragm during respiratory movements.

The valves of the heart lie on a line joining points A and C (plane of the coronary sulcus)

Mark and name each valve on that line and then use a stethoscope to listen to the each of the valves at the following points. For each valve draw an arrow from the location of the valve to point where you listen:

Tricuspid valve - 5\textsuperscript{th} intercostal space just right of sternum

Aortic - 2\textsuperscript{nd} intercostal space just right of sternum

Mitral valve - 5\textsuperscript{th} intercostal space 2cm left of sternum

Pulmonary valve - 2\textsuperscript{nd} intercostal space just left of sternum

You can also mark the surface projections of the major vessels:

IVC – point A
SVC and right brachiocephalic vein – a line from point B to the head of the right clavicle

The subclavian veins are behind the medial half of the clavicle

The left brachiocephalic vein goes from the left clavicular head across the upper half of the manubrium to join the right/SVC.

The ascending aorta goes from the aortic valve to the right side of the sternal angle

The aortic arch goes from there to the 2nd costal cartilage left of sternum and crosses the lower half of the manubrium

The descending thoracic aorta goes from there to the 12th thoracic vertebra marked earlier.

The pulmonary arteries are in the middle mediastinum and thus lie below the level of the sternal angle (higher on the left than right)

DRAW the heart and great vessels on the diagram of the thoracic cage

Indicate where you would place a Stethoscope to listen to each of the valves

NOTE: This laboratory is also a chance for you to get used to dealing with stranger’s bodies in a professional manner, you should put your “patient” at their ease and tell them what to do in a firm but friendly manner.
Count the ribs by placing fingers next to the sternum where the bones are most superficial in even the most obese or busty patient.
CARDIOVASCULAR MODULE

Physiology Laboratory

Friday, Week 3

ECG, Pulse waves, and heart sounds
ECG, pulse waves, and heart sounds

**NOTE:** For this class you need to wear loose non-constrictive clothing e.g. shorts and t-shirt or short-sleeved button-up shirt. If you wear constrictive long sleeves, long pants or jeans you will not be able to make the necessary observations. Work in groups of 3.

**BACKGROUND**

The Cardiac Pump

The beating of the heart is accompanied by both electrical activity and sound. The pattern of electrical activity produced by each heart beat cycle is called the electrocardiogram or ECG. Cardiac contractions are not dependent upon a nerve supply. A group of specialised muscle cells (called the sinoatrial node, or SA node) acts as the pacemaker for the heart. These cells rhythmically produce action potentials that spread through the fibres of the atria. The resulting contraction pushes blood into the ventricles. The only electrical connection between the atria and the ventricles is via the atrioventricular (AV) node. The action potential spreads slowly through the AV node (thus giving a time delay for ventricular filling) and then rapidly through the AV bundle and Purkinje fibres to excite contractions of both ventricles. The action potentials recorded from atrial and ventricular fibres are different from those recorded from nerves and skeletal muscle. The cardiac action potential is composed of three phases: a rapid depolarisation, a plateau depolarisation (which is very obvious in ventricular fibres), and a repolarisation back to resting membrane potential. The combined electrical activity of the different myocardial cells produces electrical currents that spread through the body fluids. These currents are large enough to be detected by recording electrodes placed on the skin. The regular pattern of peaks produced by each heart beat cycle is called the electrocardiogram or ECG (Figure 1).

![Figure 1. The general features of the ECG](image)

The components of the ECG can be correlated with the electrical activity of the atrial and ventricular muscle:

- the P-wave is produced by atrial depolarisation
- the QRS complex is produced by ventricular depolarisation; atrial repolarisation also occurs during this time
- the T-wave is produced by ventricular repolarisation.

Each side of the heart has two valves, that convert rhythmic contractions into a unidirectional pumping. The valves close automatically whenever there is a pressure difference across the valve that would cause backflow of blood. Closure gives rise to audible vibrations (heart sounds). Atrioventricular (AV) valves...
between the atrium and ventricle on each side of the heart prevent backflow from ventricles to atria. Semilunar valves are located between the ventricle and the artery on each side of the heart, and prevent backflow of blood from artery to ventricle. The cardiac cycle involves a coordinated sequential contraction of the atria and the ventricles.

The characteristic sound produced by the heart is usually referred to as a ‘lub-dup’ sound. The lower-pitched ‘lub’ sound occurs during the early phase of ventricular contraction and is produced by closing of the atrioventricular valves (the mitral valve and tricuspid valve), which prevent blood from flowing back into the atria. When the ventricles relax, the blood pressure drops below that in the artery and the semilunar valves (aortic and pulmonary) close, producing the higher-pitched ‘dup’ sound.

OBJECTIVES
In this class you will make measurements on members of your group in order to increase your understanding of the relationships between the electrical and mechanical events that enable the heart to pump blood to the various tissues of the body.

EXERCISE 1. The ECG
Setting Up The Experiment

This general set-up is used for all the exercises in this experiment. The PowerLab/4st should already be connected to your computer and turned on. The other supplied equipment required for these exercises is:

- the Bio Amp cable (using three leads)
- three electrodes to go on the ends of the leads
- A stethoscope.

1. The student volunteering for the experiment should remove any watch, jewellery, and so on from his or her wrists and ankles.
2. Plug the Bio Amp cable into the Bio Amp socket on the PowerLab unit (see Figure 2).
3. Connect the leads to Earth, CH1 negative, and CH1 positive, on the Bio Amp cable. Connect the electrodes to the leads. The disposable adhesive electrodes have electrode gel on them already.
4. Attach the electrodes to the student volunteer. Attach the black electrode to the left upper arm, the white electrode to the right upper arm, and the green electrode to either wrist. The electrodes should not be placed over the major muscles of the upper arm (that is, the biceps or triceps) because muscle activity interferes with the signal recorded from the heart. Attach the electrodes on the outer side of the arm, midway between the elbow and the shoulder.
5. Ensure that the volunteer is relaxed and sits as still as possible to minimise any signal from movement (with hands in lap, say).
Figure 2 – BioAmp cable and connection to the PowerLab for recording of the ECG

Starting the software
1. Locate and open the IMED1100 folder.
2. Open the file named ‘Ex 2 – ECG’ to open Chart. After a short time, Chart will open and the Chart window will appear on the computer screen. Channel 2 should be labelled ‘Event’ and Channel 3 should be labelled ‘ECG’. Channel 1 has been hidden at the top of the window.
3. Choose the Bio Amp … command from the Channel 3 (ECG) Channel Function pop-up menu. Observe the signal.
4. If the ECG cannot be seen, check that all three electrodes are correctly attached. Adjust the range if necessary. If the signal is noisy and indistinct, make sure that the volunteer is relaxed.
5. With the volunteer sitting quietly, click the ‘Start’ button. When you have a suitable trace, type ‘Resting ECG,’ and the volunteer’s name, and press the ‘Add’ button or Enter key to enter the comment. There should be a flat line in Channel 2 at this stage.
6. Click the Stop button to stop Chart recording. If you are saving your files, choose Save from the File menu to save the recording.

Analysis
1. Scroll through your data and observe the regularly occurring ECG cycles.
2. Use the View buttons in the Chart window to set the horizontal compression to 1:1.
3. NOTE the small ‘M’ with a triangle under it in the lower right hand corner of the screen, and two numbers, one near the top right of the screen (showing time), and another on the top right of each channel (showing the value of the trace at the cursor position). When the ‘M’ marker is clicked and placed anywhere on a trace, the numbers change from ‘xx.xx’ to ‘Δxx.xx’. The ‘Δ’ (delta) symbol indicates that the value is the difference between the position of the Marker and Waveform Cursor, and now show the difference in the value (time or voltage) at the current position of the cursor from the value at ‘M’. This utility is very useful for measuring amplitudes and times.
4. Measure the amplitudes of four P waves, four QRS complexes, and four T waves from the ECG trace. Place the ‘M’ at the base of a wave and move the Waveform Cursor to the peak of the wave. Then
read off the amplitude value from the Rang/Amplitude display directly above the ‘ECG’ channel title. Record the average amplitude in a table.

5. Using the Marker and Waveform Cursor, measure the durations of four P waves, four QRS complexes, and four T waves from the ECG trace. Place the Marker on the ECG trace immediately before the wave of interest, move the Waveform Cursor to the end of the waveform, and read off the time from the Rate/Time display. Calculate the average duration for each waveform from four separate ECG cycles and record your results.

6. Use the View buttons in the Chart window to compress the view horizontally to 10:1. Place the marker before a QRS complex and move the waveform cursor to the right until roughly a 15-second difference is shown in the Rate/Time display (this should appear as ‘Δ15s’). Count the number of QRS complexes between the Marker and the Waveform Cursor. Multiply this number by four to calculate resting heart rate in beats per minute (bpm).

6. Use the View buttons in the Chart window to set the horizontal compression to 2:1. Measure the time interval (in seconds) between three pairs of adjacent R waves using the Marker and Waveform Cursor. For each interval, calculate the heart rate as follows:

\[
\text{Rate} = \frac{60}{R-R \text{ interval}} \quad \text{(beats/min)}
\]

Questions

What can you say about the amplitude of the various waves in different cardiac cycles? Why is the amplitude less than that of a ventricular myocardial cell?

The P wave and the QRS complex represent depolarisation of the atrial and ventricular muscle respectively. Why does the QRS complex have the largest amplitude?

In step 5 you calculated a heart rate averaged over 15 seconds whereas in step 6 you calculated ‘instantaneous’ heart rates. Is there a difference between these values? If so, can you relate the differences to the phases of respiration?

The range for a normal resting heart rate is 60 to 90 bpm. A trained athlete could have a resting heart rate of 45 to 60 bpm. Why might a very fit person have a slower heart rate than someone of average fitness?

Are the amplitudes and durations of the various waves in different individuals similar or very different?

What differences in heart rate did you observe between individuals? Is there any correlation between heart rate and gender or apparent fitness?
Half of the computers in the lab will be set up for Exercise 2, and half for exercise 3. When you finish Exercise 1, move directly to the next Exercise on your computer. By the time you have finished, other groups will be finishing the alternate exercise, and you can then move to that computer to complete the alternate exercise.

EXERCISE 2: ECG and heart sounds

Objectives
To measure and correlate the ECG and heart sounds in a resting volunteer using a stethoscope and also use the chest microphone which should be plugged in to channel 2.

Your tutor will demonstrate how to use the stethoscope and the microphone.

Procedure
The stethoscope bell is better than the diaphragm for this exercise because it blocks off room noise. It helps for everyone to keep the noise down. The bell (the cup-shaped end) of the stethoscope should be placed directly on the skin over the heart to hear the heart sounds clearly. Run the ECG trace whilst listening and try to place the heart sounds in relation to the electrical events.

The volunteer should place the bell of the stethoscope on the left side of his or her chest, using the right hand. (It is easy enough to do this under one’s shirt.) The stethoscope should be moved to different positions until clear heart sounds are heard by the student listening to the stethoscope. The sounds are soft, and room noise must be kept low. Once clear heart sounds are heard, the volunteer should hold the stethoscope in place with the right hand while the student listening to the stethoscope listens and records.

Now use the chest microphone to synchronise the sound events more accurately than you can do by listening. This is not easy in all subjects and takes some trial and error with the placement of the microphone on the chest.

Analysis
1. Select a region of data with two or three cardiac cycles, by dragging in the Time axis area (this will select both of the displayed channels).
2. Select Zoom Window from the Windows menu. The Zoom window appears with the Event (i.e. the microphone signals and ECG signals overlaid.
3. Note the correlation between Event and ECG signals.
4. Note any differences from the expected timing of the Event signal.

QUESTIONS
Explain why ventricular contraction (systole) and the ‘lub’ sound occur immediately after the QRS complex. Explain why ventricular relaxation (diastole) and the ‘dup’ sound occur after the T wave.
EXERCISE 3:
ECG & arterial pulse

The beating of the heart results in a blood flow that is pulsatile. In this exercise you will measure the pulsatile flow of blood through the finger of a student volunteer and correlate it with the ECG. In addition you will palpate various arteries.

Background

The arterial system functions as a pressure reservoir. Blood leaves the arterial system continuously through the capillaries, but enters intermittently from the heart. When the ventricles contract (called ‘systole’), the semilunar valves open and blood passes into the arterial system. At this point the arteries expand and the blood pressure increases. The peak value is called the systolic pressure. The ventricles then relax (called ‘diastole’), and fill with blood from the veins, ready for the next systole. During diastole, blood flows out of the arterial system through the capillaries and the arterial pressure decreases. When the arterial blood pressure is at its lowest — immediately before the contracting ventricle pushes blood into the arteries — this value is called the diastolic pressure. Although the variation in arterial blood pressure during the cardiac cycle is smoothed out by the inherent elasticity of the major arteries, blood still exhibits pulsatile flow through the smaller arteries.

Setting up the experiment

1. The ECG leads should be set up as before.
2. Place the pressure pad of the finger pulse transducer against the distal segment (the tip) of the middle finger of either hand of the volunteer. Use the Velcro strap to attach it firmly — neither loose nor tight.

Starting the software

1. Set up as before.
   Note: Channel 2 is the raw signal from the finger pulse transducer and is an indication of the net rate of blood flow into the finger pulp. The time integral of Channel 2 is displayed on Channel 1, and gives an idea of the change in finger pulp volume over time.
3. You are now ready to begin the exercises.

Procedure

1. Choose the Bio Amp… command from the Channel 3 (ECG) Channel Function pop-up menu. Observe the signal. If the ECG cannot be seen, check that all electrodes are correctly attached. If the signal is noisy and indistinct, make sure that the volunteer is relaxed.
2. Click the OK button to return to the Chart window.
3. Choose the Input Amp… command from the Channel 2 (Blood Flow) Channel Function pop-up menu. Adjust the value in the Range pop-up menu of the dialog box that appears so that the signal occupies about a half to two thirds of full scale when the volunteer has both hands in his or her lap. You may need to adjust the signal in Channel 1 (Vol. Pulse) as well.
4. Click the OK button to return to the Chart window.
5. Click the Start button, and record for about ten seconds.
6. Click the Stop button to stop Chart recording.
7. If you are saving your files, choose Save from the File menu to save the recording.

**Analysis**

1. Drag the Marker to the peak of a QRS complex in Channel 3.
2. Move the pointer to the right, so that the Waveform Cursor in Channel 2 (Blood Flow) lies on the following peak of the blood flow trace.
3. Note the time difference, Δt, from the Rate/Time display at the top right of the Chart window. This is the time between the two events.

**QUESTIONS**

What produces the QRS complex in the ECG?
What does the peak in the blood flow trace represent?
Which processes take place between these two events?
Does the falling phase of the volume pulse have a small, transient plateau or upward deflection? This is called a dicrotic notch. What causes the dicrotic notch?

---

**Palpation of arterial pulses (this can be done in your own time at home)**

**Objectives**

To palpate various pulses (that is, to examine them by the sense of touch and pressure), and to attempt to understand what is involved in the pulse.

**Procedure**

No special equipment is required for this exercise. Each student should try to palpate the pulses on another student.

1. Feel the subject’s radial pulse, at the wrist. You should feel it with the first three fingers, your index, middle, and ring fingers, placed in a line along the length of the radial artery.
2. Don’t use your thumb for palpation — it has a strong pulse in it, and you may end up feeling your own pulse instead of the subject’s. Also, don’t press too hard — only light to moderate pressure is needed to feel a pulse.
3. Feel the brachial artery pulse at the elbow. When looking for the arteries, it is best to glide the fingers back and forth slowly over the region rather than immediately applying pressure.
4. Feel the dorsalis pedis pulse between the first and second metatarsal bones on the dorsum of the
foot.
5. Feel the posterior tibial pulse, inferior to the medial malleolus.
6. Feel the carotid pulse in the neck, by placing the index, middle, and ring fingers in a line and pressing gently to one side of the trachea. The carotid pulse is very strong and easily palpated.
7. Feel the facial pulse. It is a delicate pulse requiring skill to discern. The facial artery passes from the neck towards the face, by winding around the edge of the mandible about 1–2 cm in front of the angle of the mandible.

QUESTIONS
When you feel a pulse, do you feel (a) the blood flow or (b) the pressure wave. (Note: the transmission of a pressure wave does not indicate flow. Think of the analogy of transmission of sound waves in air – the sound waves do not correlate with flow of air from the source of the sound to the receiver).
Anatomical sites where a pulse can be palpated often correspond to ‘pressure points’ for stopping haemorrhage in first-aid treatment. Why?
1) Why is the amplitude of the waves in the ECG less than that of the amplitude of a depolarisation in ventricular myocardial cell?

2) The P wave and the QRS complex represent depolarisation of the atrial and ventricular muscle respectively. Why does the QRS complex have the largest amplitude?
3) In step 5 you calculated a heart rate averaged over 15 seconds whereas in step 6 you calculated ‘instantaneous’ heart rates. Is there a difference between these values? If so, can you relate the differences to the phases of respiration?

4) The range for a normal resting heart rate is 60 to 90 bpm. A trained athlete can have a resting heart rate of 45 to 60 bpm. Why might a very fit person have a slower heart rate than someone of average fitness?

5) What differences in heart rate did you observe between individuals? Is there any correlation between heart rate and gender or apparent fitness?

6) Explain why ventricular contraction (systole) and the ‘lub’ sound occur immediately after the QRS complex.
7) Explain why ventricular relaxation (diastole) and the 'dup' sound occur after the T wave.

8) What produces the QRS complex in the ECG?

9) What does the peak in the blood flow trace represent?

10) Which processes take place between the QRS and the peak in the blood flow trace?

11) Look carefully at the volume pulse trace: you should see a small, transient plateau or upward deflection? This is called a dicrotic notch. What causes the dicrotic notch?
12) When you feel a pulse, do you feel (a) the blood flow or (b) the pressure wave?

13) Anatomical sites where a pulse can be palpated often correspond to ‘pressure points’ for stopping haemorrhage in first-aid treatment. Why?
CARDIOVASCULAR MODULE
Anatomy Laboratory

Wednesday, Week 4

Histology of the cardiovascular system
Material used in this Laboratory:

Histology Lab microscopes

Permission to log on to histology system
NS 100  Cardiovascular system

Laboratory class: Histology of the cardiovascular system

Read and study these notes and use your textbook to become familiar with the contents BEFORE you come to the lab. This laboratory will take place in the histology laboratory (across the corridor from the dissecting room).

Outcomes

1. The student will be able to describe the basic microscopic structure of the cardiovascular system in terms of the characteristic tunica intima, media and adventitia components.
2. The student will be able to identify the different kinds and blood vessels and relate their structure and function.
3. The student will be able to recognise the structure of cardiac muscle and relate its special features to its function.
4. The student will be able to identify lymphatic tissues and understand their basic function.

STRUCTURE OF BLOOD VESSELS

1. Elastic artery

Examine the slide of the elastic artery. Draw a small diagram and label the three tunics

Tunica intima
Tunica media
Tunica adventitia

What sort of epithelium is seen in the tunica intima lining the lumen?

What special name is given to this epithelium?

If the section is stained for elastin, you will see one of the tunics completely filled with elastic tissue. What is the function of this tissue here?

Describe the structure of the tunica adventitia

Name 3 elastic arteries
2. Muscular artery

Examine the slide of the muscular artery. Draw a small diagram and label the three tunics
Tunica intima
Tunica media
Tunica adventitia

In this kind of artery the elastic tissue is mainly found at the inner and outer edges of the tunica media. What are these zones called?

What kinds of tissue fills the tunica media
Name 3 arteries of this sort

3. Small artery (arteriole)

Study the slide with small arteries. Can you identify the 3 tunics of a small artery? If so what tissues comprise each tunic

How is the muscle layer arranged in these small arteries (arterioles)?

What functional role does this serve?
Where are small arteries (arterioles) found?
What is the function of small arteries (arterioles)?
4. Capillaries
Examine the capillary preparation. Which layers/tunics are found in a capillary

Name the three (morphological) types of capillaries and specific tissues/organs in which each type occurs.

For what specific functional role/s are each type of capillary specialised?

5. Veins
Study the slide with the large vein. And describe the structures found in each layer/tunic

What is the function of such large veins? and how do the structures seen relate to this

Observe the demonstration slide/image of valves in veins and review their function.
CARDIAC MUSCLE

1. Examine the slide of Cardiac muscle and complete the table below that summarises the differences between the three types of muscle in the body.

<table>
<thead>
<tr>
<th></th>
<th>Smooth</th>
<th>Voluntary</th>
<th>Cardiac</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multicellular fibres</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shape of fibres</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Position of nuclei</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Special features</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

What structural components of junctional complexes comprise the intercalated disc?

____________________________________

Name two functions for intercalated discs that are directly related to the function of cardiac muscle

____________________________________

If your slide shows the inner and outer surfaces of the section of heart muscle (myocardium), you may be able to see the simple epithelium that lines the inner aspect of the heart chambers (what is this layer called?)

____________________________________

And the layer that coats the outer aspect of the heart?

____________________________________

What is the most notable difference between the myocardium of the atrium and that of the ventricles?

____________________________________

What is the functional significance of this difference?

____________________________________

What does a heart valve consist of histologically?

____________________________________
LYMPHATIC ORGANS

1. Lymph node

Examine the section of a lymph node, draw a diagram (low power) and describe the main types present and the function of the two main regions (ie. cortex and medulla).

Afferent lymphatic
Capsule
Subcapsular space
Cortex
Lymph nodules
Medulla and medullary cords
Afferent arteriole
Efferent lymphatic
Paracortex
Cortex

Medulla


2. Spleen

Examine the section of the spleen. Identify the thick capsule and trabeculae, and regions called “red pulp” and “white pulp”

For each of those regions indicate the main types of cells present and the main functions

White pulp


Red pulp


Where is the central artery?

How does blood flow through the spleen?

Relate this to the function of the spleen
From your examination of the lymph node and spleen, describe the basic cell types that characterise lymphatic tissues

3. Lymphatic vessel. See the demonstration slide/image of the histological structure of a large lymphatic vessel.

Name some other lymphatic organs

THESE WILL BE DEALT WITH IN THE SUBSEQUENT PARTS OF THE COURSE
CARDIOVASCULAR MODULE
Physiology Laboratory

Friday, Week 4

SYMBIOSIS: Cardiac function
Symbiosis: Cardiac function

BACKGROUND

The Cardiac Pressure-Volume Relationship

The heart functions as a mechanical pump. Each time the heart contracts, it applies pressure to the blood contained within its chambers. In cardiac function, the most important variables are the pressures generated inside the atria and ventricles, and the volumes contained within them. If the valves are functioning properly, this pressure causes blood to flow through the heart in a single direction. The most important chamber is the left ventricle: it does far more work than the right ventricle or either atrium. We will focus on the pressure-volume relationship for the left ventricle, although the basic concepts also apply to the right ventricle and the atria.

VENTRICULAR PRESSURE-VOLUME RELATIONSHIPS

The diagram above shows the ventricular chamber pressure as a function of the volume contained within the ventricle. At the end of diastole, the ventricle is analogous to a passive elastic bag. As the ventricular volume increases, so does the ventricular pressure. The relationship of pressure to volume in the relaxed state is termed passive or end-diastolic pressure-volume curve. When the ventricle contracts (entering the systolic phase), the pressure-volume relationship becomes much steeper, and the ventricle generates higher pressure at the same volume. End-systole is defined as the point in time when the pressure-volume relationship is the steepest. The relationship of pressure to volume in the maximally contracted state defines the active or end-systolic pressure-volume curve. Thereafter, the ventricle begins to relax and the pressure-volume relationship returns back to the passive state. In the following discussion, we will focus on two specific states and the corresponding pressure-volume relationships: the passive (end-diastolic) state and the active (end-systolic) state. A cardiac cycle can be thought of as a cyclic transition between these two states. Ventricular systole is comprised of isovolumic contraction and ejection phases. Ventricular diastole consists of isovolumic relaxation and filling.
Several events can be noted on the pressure volume loop. The stroke volume (SV) is the width of the loop; end-systolic volume (ESV) and end-diastolic volume (EDV) are shown on the horizontal axis. The aortic valve opens when the ventricular pressure exceeds the aortic (diastolic) pressure. The peak systolic ventricular pressure is at the very top of the PV loop - pressure typically falls a bit before the end-systolic pressure volume curve is reached. Provided that the aortic valve is normal, left ventricular and aortic pressures during ejection are nearly equal. The end of systole occurs when the ventricle has contracted as far as it can - to the end-systolic pressure-volume relationship. When the ventricle stops contracting, flow stops, and the aortic valve closes. The ventricle then relaxes back to the passive curve.

If you know the cardiac output, it is easy to calculate the stroke volume, because stroke volume is just cardiac output divided by heart rate. Stroke volume can also be calculated from the ventricular end-diastolic and end-systolic volumes:

\[ SV = EDV - ESV \]

A calculated parameter, the ejection fraction (EF), is often used as a clinical measure of the efficiency of ventricular contraction. The ejection fraction is defined as the ratio of stroke volume to left ventricular end-diastolic volume:

\[ EF = \frac{SV}{EDV} \]

In clinical medicine, instantaneous measurements of ventricular volumes are difficult. Several methods are possible, e.g., nuclear scanning, echocardiography, fast computerized tomography, magnetic resonance imaging, and cardiac angiography. Sophisticated techniques are necessary to obtain accurate quantitative estimates of volume; otherwise the results should usually be viewed as approximate.

In this class you will use the SymBioSys program to explore the intrinsic physiological regulation of cardiac function. The exercises are designed to help you understand the relevance of preload, afterload, contractility and compliance in determining cardiac performance. You have used the program in a previous laboratory class, so you should know your way around the screens. Go to the upper left window and open “The Circulatory System” Window. Then open “8 - Cardiac Performance”, and next open the “Exercises Folder”.

- From the exercises that are listed open the second one “Cardiac Preload”. Work through this exercise and record your findings in your notebooks.
PRELOAD

Preload is one of the major determinants of ventricular contraction and consequently, of stroke volume and cardiac output. Historically, the term preload originated from isolated muscle experiments, referring to the tension used to stretch a resting muscle before active contraction began. Contractile force increases as the resting muscle (sarcomere) length is increased, up to a maximum length. The same phenomenon occurs in the intact ventricle, but resting muscle tension cannot be readily measured in the intact ventricle and it varies with the location in the heart. In the intact ventricle, preload is quantified as the ventricular volume prior to contraction: the end-diastolic volume (EDV). Acute changes in preload in an individual subject are best characterized by changes in EDV. The strength of ventricular contraction is greater when the preload is higher. We will focus on filling of the heart during diastole. Blood flows into the ventricle when the heart is relaxed, so the volume increases along the diastolic (passive) pressure-volume curve. The more blood flows into the heart, the higher the pressure even before contraction. Diastole ends when the heart begins to contract, closing the A-V valves and isolating the ventricle from the rest of the vascular system. EDV and EDP (end-diastolic pressure) are defined by this point.

Preload variations are probably the most important regulators of cardiac output under normal circumstances. As discussed above, EDV is the best descriptor of preload, but is difficult to quantify precisely in vivo. EDP is a surrogate measure, and is non-linearly related to EDV (according to the diastolic [passive] pressure-volume relationship). The strength of atrial active contraction can modulate ventricular preload. The terminal contraction of the atrium serves primarily to augment ventricular preload prior to ejection.

NOW WORK THROUGH THE EXERCISE. DO NOT PRINT OUT THE CHARTS FROM WITHIN THE PROGRAM BUT INSTEAD MAKE YOUR OWN CHARTS IN YOUR NOTEBOOKS AS REQUIRED.

RECORD THE CHANGES THAT OCCUR WHEN BLOOD WAS INFUSED.

Question: Describe what happened when you infused blood, and why it happened.

Question: In the exercise you infused blood to increase total blood volume and preload. How else could preload be increased?

AFTERLOAD

Like preload, the term afterload originated from isolated muscle experiments. It was defined as the muscle tension during the shortening phase of muscle contraction. This tension was often held constant at a prescribed level, resulting in isotonic muscle shortening. The extent and rate of muscle shortening were reduced as afterload was increased. The same phenomenon occurs in the intact ventricle. However, muscle tension cannot be readily measured in the intact ventricle. Moreover, it varies with the location in the heart and the time-point in the cardiac cycle.
Instantaneous left ventricular pressure during ejection is an index of left ventricular afterload. A primary determinant of the left ventricular pressure during ejection is the arterial resistance. When contracting against a high arterial resistance, a left ventricle will eject less blood than when it is contracting against a lower resistance. Another significant factor is the compliance of the aorta; pressure will rise more rapidly when the ventricle is ejecting into a stiffer aorta. Thus, interaction of the ventricle with properties of the arterial circulation determines how the left ventricular pressure changes during ejection. Although instantaneous left ventricular pressure during ejection is perhaps the best measure of left ventricular afterload, the extent of ventricular ejection is determined by ventricular end-systolic pressure (ESP). Thus, as a determinant of stroke volume, end-systolic pressure is a reasonably good measure of ventricular afterload.

The effects of changes in afterload on ventricular mechanical function can be understood in the context of the pressure-volume loop when other determinants of ventricular function (preload, heart rate, and contractility) are held constant.

![Pressure-Volume Loop](image)

It is easily seen that when afterload is low, the ventricle can eject to a much lower end-systolic volume, given the same preload and contractility. Accordingly, both stroke volume and ejection fraction are much higher when afterload is low. This principle is the basis for the most effective known therapy for congestive heart failure. Drugs which cause long-term vasodilation (angiotensin converting enzyme inhibitors in particular) lower afterload, improve stroke volume, improve cardiac output, and improve long-term survival in heart failure.

- Open Exercise 3. Afterload (remember to reset before you start a new exercise)

In the exercise, afterload is varied by infusing nitroprusside which lowers arterial BP by relaxing resistance vessels. Now work through the exercise and note the responses as you proceed. Set the infusion rate etc as in your did previously. Expand the window to enhance your view. Watch the changes in the PV loop during the infusion.

**NOW WORK THROUGH THE EXERCISE. DO NOT PRINT OUT THE CHARTS FROM WITHIN THE PROGRAM BUT INSTEAD MAKE YOUR OWN CHARTS IN YOUR NOTEBOOKS AS REQUIRED**

Record and describe the changes that occur when nitroprusside is infused.

**Question** Why does LVESV change?
Question: Why does cardiac output change?

**CONTRACTILITY**

Ventricular contractility is the intrinsic potential of the ventricle for generating pressure and/or flow. One of the indices of ventricular contractility is the peak active pressure-volume relationship, the end-systolic pressure-volume relationship. The more active pressure the heart can generate at a given volume, the greater the contractility. The effect of an isolated decrease in contractility can be seen in the following pressure-volume diagram, where a decrease in contractility is seen as a change in the slope of the end-systolic pressure-volume relationship. Note that preload, afterload, and heart rate are assumed to be unchanged.

Preload is the same for the two loops, so the ventricle begins contraction from the same end-diastolic volume. Also the afterload is the same, so that the end-systolic pressure is the same for the two contractions. Yet the end-systolic volumes are markedly different, because the ventricle stops ejecting at a much larger volume for the contraction with lower contractility. Thus, the stroke volume (or equivalently, cardiac output since heart rate is fixed) decreases as ventricular contractility falls. In an intact cardiovascular system, physiologic responses will alter preload and afterload in response to the decreased contractility such that there is an attempt to normalize ventricular function. For example, preload will increase following a decrease in ventricular contractility. This physiology is typical of patients with congestive heart failure.

Sympathetic stimulation increases contractility while hypoxia and acidosis decrease contractility. In humans, the most common cause of impaired contractility is cardiac muscle damage due to myocardial infarction, where portions of the ventricular wall die from lack of blood supply.

**HEART RATE**

Physiological situations such as exercise demand large changes in cardiac output. Athletes can increase cardiac output by a factor of 5 or more, yet stroke volume can only increase by about 30% (because of increased contractility and decreased afterload). To provide a large increase in cardiac output the heart rate, the number of stroke volumes ejected per minute, must also increase. During exercise, the increase in cardiac output is made up by a combination of an increase in heart rate and a small increase in stroke volume.

Increased rates are associated with shorter durations of diastole, which can reduce ventricular filling time. This becomes problematic only at extremely high heart rates or in situations where ventricular filling is otherwise limited (as in mitral stenosis). Thus, as heart rate increases, cardiac output increases at first, then
eventually decreases. The heart rate which maximizes cardiac output varies between individuals, but is usually taken to be about 180 bpm.

- **Open Exercise 6, Heart Rate (remember to reset before you start a new exercise)**

**NOW WORK THROUGH THE EXERCISE. DO NOT PRINT OUT THE CHARTS FROM WITHIN THE PROGRAM BUT INSTEAD MAKE YOUR OWN CHARTS IN YOUR NOTEBOOKS AS REQUIRED**

In the exercise, you manipulate the heart rate to find how the circulation responds. Work through the exercise and note the responses as you proceed.

You will be required to determine the HR at which CO is a maximum. Think about how many data points (manipulate HR and measure CO) you need to be sure you have attained the peak value for CO.

Expand the window to enhance your view. Watch the changes in the PV loop as you change the heart rate. (Note that this is **NOT** an accurate model for what happens in exercise, when there are simultaneously large increases in myocardial contractility and substantial decreases in afterload due to vasodilation in the working muscles).

**RECORD THE CHANGES THAT OCCUR WHEN HEART RATE WAS CHANGED.**

**Question** Why does the CO not double when HR is doubled?

**Question** Why do LVEDV and LVESV change?

**Question** What is the HR when CO is at a maximum?

- Now you need to access the DRUG & FLUID INFUSOR panel as in a previous exercise, and select epinephrine and norepinephrine at default rates. RESET again and infuse epinephrine (adrenaline) at 2 µg/kg/min, and record the responses. Then RESET again and infuse norepinephrine (noradrenaline) at 2 µg/kg/min.

**DESCRIBE THE RESPONSES OF THE TWO INFUSIONS AND COMPARE THE EFFECTS ON CONTRACTILITY, HEART RATE, SVR AND CO.**

**Question** What is the difference in afterload with the two infusions?

**Question** How do the responses to epinephrine compare with the previous exercise when only HR was increased?
Table 1: Preload

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>After blood infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV Contractility</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sinus rate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVESV</td>
<td></td>
<td></td>
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<tr>
<td>SV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (observed)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SVR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic Pressure (mean)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1) Preload exercise: Describe what happened when you infused whole blood and explain why these things happened.
2) Apart from infusing whole blood, how else can preload be increased?

### Table 2: Afterload

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>After nitroprusside infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV Contractility</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sinus rate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV</td>
<td></td>
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<tr>
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<td>CO</td>
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<tr>
<td>HR (observed)</td>
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<tr>
<td>SVR</td>
<td></td>
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<tr>
<td>Aortic Pressure (mean)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

3) Afterload exercise: Why does LVESV change after you infuse nitroprusside?
4) Afterload exercise: Why does CO change after you infuse nitroprusside?

Table 3: Sinus rate variance

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Sinus rate 105</th>
<th>Sinus rate 140</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV Contractility</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Sinus rate</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>LVEDV</td>
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<td>LVESV</td>
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<td>CO</td>
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<tr>
<td>HR (observed)</td>
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<tr>
<td>SVR</td>
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<tr>
<td>Aortic Pressure (mean)</td>
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</tbody>
</table>

5) Provide a plot showing clearly what happened to CO when you varied HR. (NOTE: Are three points enough to determine the relationship?)
6) In the exercise where you varied HR, why does CO not double when you double HR?

7) In the exercise where you varied HR, why do LVEDV and ESV change?

8) In the exercise where you varied HR, what is the HR when CO is at a maximum?
Table 4: Effects of sympathetic agonists

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Epinephrine (adrenaline)</th>
<th>Norepinephrine (noradrenaline)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV Contractility</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sinus rate</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>LVEDV</td>
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<tr>
<td>LVESV</td>
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<td>SV</td>
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<tr>
<td>CO</td>
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<tr>
<td>HR (observed)</td>
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</tr>
<tr>
<td>SVR</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic Pressure (mean)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

9) Is there a difference in afterload after you infuse epinephrine (adrenalin) as opposed to norepinephrine (noradrenalin)? Explain why or why not.

10) How do the responses to norpinephrine compare to the previous exercise when only HR was increased?
CARDIOVASCULAR MODULE
Anatomy Laboratory

Wednesday, Week 5

Blood vessels of the abdomen and pelvis
Material used in this Laboratory:

Two sets of four tables

STATION 1: Abdominal Aorta & Its Branches – prossections, Xrays, CT and angiograms

STATION 2: Veins of the Abdomen – abdominal prossections, venograms, spinal cord and veins, Dissected vertebral column.

STATION 3: Pelvic Blood Vessels – plastic models, pelvic prossections

STATION 4: Portocaval Anastomoses. Angiograms, venograms, large light box, plastic model of the back.
NS 100  Cardiovascular system

**Laboratory class on the Blood vessels of the Torso**

Read and study these notes and use your textbook to become familiar with the contents BEFORE you come to the lab.

**Outcomes**

1. The student will be able to list the 3 categories of branches of descending aorta, and to identify the main branches on a diagram or specimen.
2. The student will be able to outline the main vessels that supply the gastrointestinal tract and to describe their patterns of anastomosis.
3. The student will be able to explain the portal system, and locate its main parts on a specimen.
4. The student will be able to list and identify the tributaries of the inferior vena cava.
5. The student will be able to outline the branches of the internal iliac artery and the corresponding venous drainage
6. The student will be able to recognise that there are no valves in any of the veins of the torso (except where major vessels enter from the limbs) and that this results in possibilities for blood travel by different routes within the torso.
Station 1 ABDOMINAL AORTA AND ITS BRANCHES

Look at prosections and angiograms of the abdominal aorta and follow it from its beginning in front of the 12\textsuperscript{th} thoracic vertebra to where it bifurcates in front of the 4\textsuperscript{th} lumbar vertebra. The aorta terminates by dividing into the left and right common iliac arteries, which soon divide again into internal and external iliac arteries. Internal iliac arteries pass into the pelvis and the external iliac arteries pass over the front of the hip bone into the anterior thigh. Can you see a small continuation of the aorta called the median sacral artery?

The abdominal aorta’s collateral branches can be grouped in three categories. Use the prosections to study the arteries listed:

1) Gastointestinal branches (coeliac, superior mesenteric, inferior mesenteric)
2) Branches to paired organs (renal, adrenal and testicular or ovarian) These all branch from the aorta near the kidneys. Can you explain why the testicular and ovarian arteries descend from there to the scrotum or pelvic cavity respectively
3) Body wall branches (inferior phrenic, and 3 lumbar arteries)

Study the deep prosections of the abdomen and also the angiograms that show the renal and body wall branches of the aorta.

**Gastointestinal arteries.**

Each of the three branches to the gastrointestinal tract has a number of branches. Use prosections to try to identify those listed:

1. **Coeliac**
   - **Common hepatic, Splenic, Left gastric**
     - **Common Hepatic** => right gastric, gastroduodenal, **Proper hepatic**
     - **Gastroduodenal** => right gastroepiploic (+ duodenal and pancreatic arteries).
     - **Proper hepatic** => **cystic and left and right hepatic**
     - **Splenic** => **Short gastric, left gastroepiploic** Left gastric => **Oesophageal**

![Diagram of the abdominal aorta and its branches](image-url)
2. **Superior mesenteric** => middle colic (MC), many jejunal and ileal branches to the small intestine, ileocolic (IC) and **right colic arteries** (RC).

3. **Inferior mesenteric** => Left colic (LC), sigmoidal branches and **superior rectal artery** (SR)

Try to find these major gastrointestinal arteries on the prosections and also on the angiograms on display.

**Anastomoses in the GIT:**
Examine the arterial supply to the stomach and note the anastomoses between the left and right gastric and the left and right gastroepiploic arteries
Examine the arteries in the mesentery of the small intestine and note the multiple anastomosing loops between branches of the ileal and jejunal arteries.
Note that the right, middle and left colic and the sigmoid arteries anastomose around the margin of the large intestine. (see figures on previous page)

Abdominal plain film X-rays, CT scans plus intravenous or intra-arterial angiograms are provided, compare these with the cadaveric anatomy.
Station 2 VEINS OF THE ABDOMEN

**Gastrointestinal veins – The portal vein**

All the blood that supplies the gastrointestinal tract returns to the liver in the portal vein. Use a prosection to find where the inferior mesenteric, superior mesenteric and splenic veins join to form the portal vein and trace it to the liver. Label the diagram below.

Also look for the hepatic veins that drain from the liver into the inferior vena cava.

Why is it called the “Portal vein”

How does this system differ from the venous drainage of other regions

What is the purpose of this system

---

**Veins of the paired organs**

Examine the IVC and follow it from its beginning where the common iliac vein unite behind the common iliac arteries, to where it pierces the diaphragm behind the liver. Note that the renal veins join the IVC in front of the plane of the aorta. Note also that because the IVC is to the right of the aorta, the left renal vein is much longer than the right and crosses the aorta. The left testicular, ovarian and suprarenal veins join the left renal vein. However the right veins each join the IVC directly.

Study the chart that shows some common variations in the inferior vena cava, and understand the embryological basis of these variations.

Also you should be aware that the veins of the abdomen and pelvis have no valves except where large veins enter from the limbs (see the figure over page which shows the nearest venous valves to the heart)
**Body wall veins**

The lumbar veins lies on the plane of the body wall and are connected by a vertical channel called the ascending lumbar vein (which also connects to the azygos system in the thorax).

**Vertebral venous plexus**

Look at preparations of the vertebral column for remnants of the network of veins that surround it (unfortunately, many of these veins are removed to display other structures). This system of veins has no valves and is connected to all the intercostal, lumbar and pelvic veins. If the IVC was compressed at the level of the liver how might blood from the abdomen and pelvis reach the heart? Suggest two pathways and use the schematic diagram provided to show these connections:
Station 3 PELVIC BLOOD VESSELS

Study the projections showing the structures on the lateral walls of the pelvic cavity. Notice that the internal iliac arteries enter the pelvic cavity and give off many branches. Those branches supply the organs of the pelvic cavity and also the body wall, perineum and lower limb. Their branching pattern is variable and frequently the branches can only be identified by following them to their destination.

The first branches:
Superior gluteal artery passes out though a hole (greater sciatic foramen) in the posterior pelvic wall – Find it in other projections showing the gluteal region.
Iliolumbar artery comes back out of the pelvis cavity and replaces the 5th lumbar artery by supplying the lower part of the abdominal wall posteriorly and the iliac fossa.
Lateral sacral arteries run down the front of the sacrum and give small branches to each sacral foramen (equivalent of segmental arteries like intercostal and lumbar)

A little further down
Superior vesical artery (a visceral branch) passes to the upper surface of the bladder, and has a fibrous continuation which is the obliterated umbilical artery.
The obturator artery goes around the lateral pelvic wall to the hole in the anterior pelvic wall (the obturator foramen). It passes through with the obturator nerve and supplies the medial part of the thigh (find it among the muscles on the medial thigh).
Most visceral branches come off together
Inferior vesical in males
Uterine and vaginal in females
Middle rectal
The last branches

Inferior gluteal artery also passes into the gluteal region through the greater sciatic foramen. Pudendal artery also goes through the greater sciatic foramen but it goes back into the lesser sciatic foramen and supplies the perineum (it has inferior rectal and perineal branches and ends as the dorsal artery of the penis or clitoris).

Pelvic veins

Generally the veins of the same names return with arteries to form internal iliac veins. However venous blood from pelvic viscera forms a plexus (network) of valveless veins around the pelvic organs. This plexus has no valves and has valveless connections with the portal vein (rectal portocaval anastomosis) and also with the vertebral venous plexus. When abdominopelvic pressure increases blood is forced out of the abdominopelvic cavity into the vertebral venous plexus and ascending lumbar vein.

What activities raise intra-abdominopelvic pressure?
Station 4 PORTOCAVAL ANASTOMOSES

There are places where veins which normal drain to the portal vein, come into close contact with veins draining directly to the IVC. These connections are normally composed of tiny capillaries, but if the portal vein is compressed or if liver blood flow is reduced due to disease, these connections can enlarge dramatically to allow blood to bypass the portal vein. Name 4 places where these connections (portocaval anastomoses) are common.

There are angiograms on display that show some of these anastomoses in a state of pathological enlargement.

Participate in a discussion with your colleagues and tutor about portocaval anastomoses, vertebral venous plexus, effects of abdominopelvic pressure.

Look at the angiograms, discuss where the portal blood can bypass the liver and return to the heart by other routes.

What is injected to produce an angiogram? ____________________________

Is it radioactive? ____________________________ (write the answer in large capitals – this WILL be in the exam).
CARDIOVASCULAR MODULE
Physiology Laboratory

Friday, Week 5

Cardiovascular changes in exercise
Cardiovascular changes in exercise

BACKGROUND
In this class you will make measurements on members of your group to increase your understanding of the changes in cardiovascular function that occur in physical exercise to supply the working muscles (including the myocardium) with increased oxygen and substrate. You will also measure the changes and composition and volume of respired air during exercise.

NOTE: For this class the exercising volunteers need to wear loose non-constrictive clothing e.g. shorts and loose t-shirt or short-sleeved button-up shirt. If you wear constrictive long sleeves, long pants or jeans your group will not be able to make the necessary measurements.

Until now you have considered the physiological functioning of the heart and circulation in a normal individual at rest. During the course of typical daily activities, the circulation needs to make adjustments to accommodate the demands of increased circulation when we do physical work or exercise. The distribution of blood to a particular organ in a subject during exercise may be very different from that seen at rest. For example, the blood flow to the skeletal muscles increases dramatically during exercise, while blood flow to the splanchnic region decreases. Furthermore, during exercise the total amount of blood flowing around the circulatory system (the cardiac output) may increase to many times its resting value.

Warning: This experiment involves exercise and an elevation of heart rate. It should not be performed by anyone with a history of cardiovascular or respiratory problems.

Setting up the experiment
The first thing to do is to select a subject (note the warning above) who is able and willing to complete the physical exertion required (not too arduous, but more than a casual stroll in the park). The overall plan it to measure the following parameters at rest and after 3 bouts of exercise of increasing intensity:

- ECG (to measure heart rate and what happens to the electrical events at increased rates)
- Blood pressure (with a sphygmanometer)
- Respiratory rate, minute volume of respiration

Each group should allocate duties so that someone records ECG, measures BP, counts respiration, etc.

NOTE: It will be very difficult to measure BP with the stethoscope in a noisy environment. Therefore it will be best if all groups do the exercise segments at the same time, so that the room is quiet when everybody is trying to make the measurements.

Before doing anything else:
1. Measure your subject’s weight and height.
2. Calculate your subject’s body surface area using the relationship:
0.007184 \times W^{0.425} \times H^{0.725} \text{ m}^2 \quad (\text{where } W = \text{ weight in Kg and } H = \text{ height in cm})

3. Now make an algebraic estimation of your subject’s cardiac output at rest, assuming that a person with a surface area of 1.73 \text{ m}^2 has a cardiac output at rest of 5 \text{ L / min}.

This same general set-up is used for most of the exercises in this experiment, although there is some disconnection and reconnection involved. These procedures have been covered earlier, so setting up should be easy.

The PowerLab/4st should already be connected up to your computer and turned on. The other supplied equipment required for these exercises is:

- the Bio Amp cable for ECG (using three leads)
- three electrodes to go on the ends of the leads
- an upper arm cuff, sphygomanometer and stethoscope for measuring BP
- a Fleisch tube for measuring respiratory flow rates

**PREPARATION**

1. The student volunteering for the experiment should remove any watch, jewellery, and so on from his or her wrists and ankles.
2. Plug the Bio Amp cable into the Bio Amp socket on the PowerLab unit.
3. Connect three leads to Earth, CH1 negative, and CH1 positive, on the Bio Amp cable. Connect the electrodes to the leads.
4. Put on the blood pressure cuff.
5. Attach the electrodes to the student volunteer as in a previous laboratory class but make sure that the BP cuff does not interfere with your electrodes. Attach the black electrode to the left upper arm, the white to the right upper arm, and the green to either wrist.
6. Ensure the volunteer is relaxed and sitting as quietly as possible to minimise any signal from movement.

**Starting the software**

1. Locate and open the Experiment Settings IMED1100 folder.
2. Open the file named ‘Exercise 1’. After a short time, Chart will open and the Chart window will appear on the computer screen. Channel 1 should be labeled ‘Flow rate’, Channel 2 ‘Flow’, and Channel 3 ‘ECG’. However you are only measuring ECG.
Procedure

Ensure that the volunteer is relaxed and stands as still as possible during measurements, to minimise any signal from movement. This is particularly important after each exercise sequence. The Bio Amp cable comes with a clip that can be used to gather and hold the cable and leads to help keep them still. All measurements need to be made firstly at rest, and later as quickly as possible at the cessation of each of the bouts of exercise.

1. Choose the Bio Amp… command from the Channel 3 (ECG) Channel Function pop-up menu. Observe the signal.
2. If the ECG cannot be seen, check that all three electrodes are correctly attached. If the signal is noisy and indistinct, make sure that the volunteer is relaxed; consider using alternative attachment positions. Adjust the Range pop-up menu value so that the signal occupies about 1/2 to 2/3 of the vertical axis.
3. Click the OK button to return to the Chart window.
4. Click the Start button, and record for about ten seconds; type ‘Resting ECG’ and press the Add button or Enter key to enter the comment.
5. Click the Stop button to stop Chart recording.
6. If you are saving your files, choose Save from the File menu to save the recording. Keep the file open for the next exercise.
7. Measure and record the systolic and diastolic blood pressure readings for your subject at rest, using a standard arm cuff and the bell of your stethoscope.
8. Connect your subject to the Fleisch tube and record a trace for a series of respirations to obtain measurements of air flow.

Analysis

1. Select a short part of the resting ECG trace, containing two or three cardiac cycles.
2. Select the Zoom Window command from the Windows menu. [Note: the P–R interval is the time from the start of the P wave to the start of the QRS complex. A more logical name would be ‘P–Q’ interval, but P–R is traditional].
3. Use the Marker and Waveform Cursor to make the following time measurements from the ECG waveform:
   - P–R time interval
   - QRS duration
   - S–T time interval
   - T–P time interval
   - R–R time interval
   - Calculate the heart rate from the R–R interval.

Repeat the above measurements using the first ‘good’ ECG traces after each bout of exercise.
The ECG, BP and respiratory measurements should be repeated on your volunteer subject:

1. At rest,

And then immediately after stepping up (and down) on the footstool provided every;

2. 3 seconds for 1.5 minutes
3. 2 seconds for 2 minutes
4. 1.5 seconds for 3 minutes, or until your subject is exhausted whichever comes FIRST.

Present your data in the following table, or something like it:

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Rest</th>
<th>Exercise 1</th>
<th>Exercise 2</th>
<th>Exercise 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>P–R interval</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>QRS duration</td>
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<td></td>
</tr>
<tr>
<td>S–T interval</td>
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<tr>
<td>T–P interval</td>
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<td></td>
</tr>
<tr>
<td>R–R interval</td>
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<td></td>
</tr>
<tr>
<td>Heart rate</td>
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<tr>
<td>Systolic BP</td>
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<tr>
<td>Diastolic BP</td>
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</tr>
<tr>
<td>Stroke volume *</td>
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</tr>
<tr>
<td>Cardiac output</td>
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<tr>
<td>Respiratory rate</td>
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<td></td>
</tr>
<tr>
<td>Respiratory minute volume</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

* at rest, calculate stroke volume from the estimated cardiac output and measured heart rate. For each bout of exercise estimate from the change in pulse pressure (assuming that there is a direct relationship between pulse pressure and stroke volume.

QUESTIONS

Question What happened to the R–R interval and the heart rate after exercise?
Question Note that the R–R interval consists of the sum of QRS, S–T, T–P, and P–R. Which of these became appreciably shorter when the heart rate increased? What are the implications of T–P?
Question What happened to blood pressure and why?
Question What happened to stroke volume and cardiac output, and why?
Question What happened to respiration, and why?
LAB REPORT: Cardiovascular changes in exercise

1) Complete the following table.

<table>
<thead>
<tr>
<th>Subject name:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject body mass (kg):</td>
</tr>
<tr>
<td>Subject height (cm):</td>
</tr>
<tr>
<td>Subject calculated body surface area (m²):</td>
</tr>
<tr>
<td>Subject estimated resting cardiac output (L/min):</td>
</tr>
</tbody>
</table>

2) Complete the table on Page 2 showing data after exercise.

3) What happened to the R-R interval and heart rate after exercise?

4) What happened to the blood pressure after exercise and why does it change from the resting level?
Table 2: Physiological parameters after exercise

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Rest</th>
<th>Exercise 1</th>
<th>Exercise 2</th>
<th>Exercise 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>P–R interval</td>
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</tr>
<tr>
<td>QRS duration</td>
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<tr>
<td>S–T interval</td>
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<tr>
<td>T–P interval</td>
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</tr>
<tr>
<td>R–R interval</td>
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<tr>
<td>Heart rate</td>
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<tr>
<td>Systolic BP</td>
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<tr>
<td>Diastolic BP</td>
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<td>Stroke volume</td>
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<td>Cardiac output</td>
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<td>Respiratory rate</td>
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<tr>
<td>Respiratory minute volume</td>
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</tr>
</tbody>
</table>
5) Note that the R–R interval consists of the sum of QRS, S–T, T–P, and P–R intervals. Obviously when the R–R interval decreases, some or all of these intervals must shorten. Did any become appreciably shorter when the heart rate increased? (You might like to construct and attach a plot showing the R-R interval across the 4 exercise levels on the x-axis and the four smaller intervals on the y-axis)

6) What are the implications of a change in the T-P interval?

7) What happened to stroke volume and cardiac output, and why?
8) What happened to respiration, and why?
CARDIOVASCULAR MODULE

Anatomy Laboratory

Wednesday, Week 6

Blood vessels of the head and neck
Material used in this Laboratory:

Two sets of four tables

Station 1: Subclavian Artery and Veins, thoracic vertebrae, vertebral artery model

Station 2: Common Carotid Artery

Station 3: Internal Carotid and Clinical Observations

Station 4: Venous Drainage of the Head Dry Skull, Dural skull
NS 100  Cardiovascular system

**Laboratory class on the Blood vessels of the head neck and shoulder**

Read and study these notes and use your textbook to become familiar with the contents BEFORE you come to the lab.

**General Aims and Objectives**

1. The student will be able to describe the distribution of blood from the branches of the aortic arch, and the venous return that collects into the superior vena cava.
2. The student will be able to list and identify the branches of the subclavian and axillary arteries and the anastomoses around the shoulder joint.
3. The student will be able to outline the blood supply to the brain from the internal carotid and vertebral arteries, and the brains venous drainage via dural venous sinuses.
4. The student will be able to list and describe the distribution of the branches of the external carotid artery.
5. The student will be able to outline the pattern of venous drainage of the head and neck, and explain key position of the retromandibular vein.
6. The student will be able to describe the locations of lymph nodes in the head, neck and axilla.
Station 1 SUBCLAVIAN ARTERY AND VEINS

The blood supply of the head, neck and upper limb emerges through the thoracic inlet as the three branches of the aortic arch 1. ______________________ 2. ______________________ 3. ______________________. The right brachiocephalic artery divides behind the right sternoclavicular joint to form the right subclavian and common carotid arteries.

SUBCLAVIAN ARTERY

The subclavian artery crosses the 1st rib behind the clavicle. It passes behind the scalenus anterior muscle and grooves the upper surface of the 1st rib. Find these structures on prosections and bones in the lab. The scalenus anterior muscle divides the subclavian artery into its 3 parts (medial to, behind and lateral to the scalenus anterior muscle)

1st part The vertebral artery passes vertically upwards and enters the foramen transversarium of the 6th cervical vertebra, and continues through those foraminae in each cervical vertebra. It takes some large curves and loops before entering the cranial cavity through the foramen magnum.

The Internal thoracic artery descends on the internal aspect of the chest wall beside the sternum and gives rise to anterior intercostal, musculophrenic and superior epigastric branches. The thyrocervical trunk ascends in the neck before dividing into:

- Inferior thyroid (why does it take such a loopy course?)
• The transverse cervical artery goes laterally around the neck and heads for the medial border of the scapula

• The suprascapular artery also goes laterally but it gets onto the posterior surface of the scapular (suprascapular notch) where it supplies the muscles on the back of the scapula.

Find these vessels in the neck and on the shoulder and back.

2\textsuperscript{nd} part Costocervical trunk gives off the deep cervical artery to the muscles of the posterior neck and the superior intercostal artery to the 1\textsuperscript{st} intercostal space.

3\textsuperscript{rd} part Usually no branches but occasionally gives off transverse cervical and suprascapular.
Station 2 COMMON CAROTID ARTERY

The common carotid artery has no side (collateral branches). It passes up the neck inside the carotid sheath, together with the internal jugular vein and the vagus nerve. Just before it reaches the hyoid bone at a level just below the mandible C3, it dilates (carotid sinus) and then bifurcates into the internal and external carotid arteries. Study these structures on prosections of the neck. Realise that the common carotid artery runs deep to a line from the sternoclavicular joint to just below and anterior to the angle of the mandible. Feel the carotid pulse at the top, then gradually feel for the pulse lower and lower in the neck. How low can you go?

Why can’t you feel the carotid pulse in the lower part of the neck?

EXTERNAL CAROTID ARTERY

The external carotid artery gives off 6 branches very soon after it begins (collateral branches) and then ascends into the infratemporal fossa (the region deep to the ramus of the mandible). It ends by dividing into the maxillary and superficial temporal arteries (terminal branches). Examine the prosections and identify the external carotid artery and its major branches

6 collateral branches of the external carotid artery

3 from the front:
The superior thyroid artery loops downward to the thyroid gland
The lingual artery passes deeply through the floor of the mouth to enter and run on the under surface of the tongue
The facial artery initially passes deep to the submandibular gland just below the body of the mandible, before hooking under the mandible and passing onto the face. You can feel its pulse where it crosses the mandible. It takes a tortuous course across the face heading towards the medial corner of the eye. Why does the facial artery follow such a tortuous/serpentine course?
3 branches from the back of the external carotid artery

The occipital artery goes backwards deep to the sternomastoid muscle. It grooves the skull just medial to the mastoid process before becoming superficial on the back of the head. Look on a prosection and then try to feel its pulse on your own head.

The posterior auricular artery also passes backwards but goes superficial to the mastoid process and supplies the pinna of the ear.

The ascending pharyngeal artery is a small deep branch which goes up along the side of the pharynx.

Terminating branches of the external carotid artery

Superficial temporal artery becomes superficial in front of the ear and spreads over the temporal region. It can sometimes be seen bulging through the skin, and its pulse can easily be felt.

The Maxillary artery runs forwards behind the ramus of the mandible and enters the pterygopalatine fossa beside the back of the nasal cavity. It also has 3 parts in relation to the lateral pterygoid muscle, and each part gives off five branches!

The first part has two important branches: the inferior alveolar which enters a canal in the mandible and supplies the lower teeth, and the middle meningeal which enters the cranial cavity through the foramen spinosum and supplies the skull and dura. Look at the inside of a dried skull and see the grooves formed by this artery on the bone – you can easily imagine how a fracture to the bone on the side of the skull can rupture this artery.

The second part has five muscular branches to the muscles of mastication.

The third part enters the pterygopalatine fossa and gives branches to the palate, nasal cavity and orbit.
**Station 3 INTERNAL CAROTID ARTERY and the blood supply of the cranial cavity**

The internal carotid artery does not give off any branches before it enters the cranial cavity through the carotid canal. You will need to find a deep dissection of the head and neck to follow the internal carotid artery to the carotid canal. At the same time note the internal jugular vein emerging from the jugular foramen. Look on a skull and a prosection of the cranial cavity to appreciate the course and direction of the jugular and carotid canals through the bone. Notice that both canals have a loopy course through the bone and comment on the role of these bends.

On the prosections of the cranial cavity you will have noticed the vertebral arteries entering the cranial cavity through the foramen magnum. Now look at a specimen of the brain with its blood supply intact and appreciate that the vertebral arteries first unite into the basilar artery, and then divide and participate in a circular anastomosis with the internal carotid arteries. This “Circle of Willis” and its branches will be studied in detail when you do the nervous system.

Look at the arrangement of blood vessels on the cerebral cortex of the brains provided. What are the territories supplied by the three main cerebral blood vessels?

Draw a diagram here:-

Compare the anatomy to the cerebral angiograms on display. Can you describe what a subtraction angiogram is? Have you ever tried this with your digital photographs?
Clinical observations

Watch your partner strain (hold your breath and push) and watch the external jugular vein bulge on the side of the neck. Observe the valve near the lower end of the vein and you may see other valves higher up. Why does this vein bulge when you strain?

Do other veins bulge when you strain? ____________________________________________

Have you ever noticed people’s veins bulging as they speak or sing?_________________{

Form into groups around each demonstrator and participate in a discussion with your colleagues and tutor about lymphatic drainage of the head, and the clinical significance of emissary veins. Look at the X rays, CAT scans and MRIs
Station 4 VENOUS DRAINAGE OF THE HEAD

Maxillary and the superficial temporal veins unite to form the retromandibular vein. Find a superficial dissection which shows the retromandibular and external jugular veins. The retromandibular vein divides near the angle of the mandible.

The posterior branch joins the posterior auricular vein to form the external jugular vein which passes superficially over the sternomastoid muscle and descends to join the subclavian vein at a point 1cm above the middle of the clavicle.

Draw a diagram to illustrate the pattern of veins described here

Find the anterior branch of the retromandibular vein see that it joins the facial vein and then goes deep to join the internal jugular vein. The facial vein drains the forehead and face. It has important valveless connections through the orbit to the cavernous sinus and also through the cheek to connect with the maxillary veins. Also look for superior and middle thyroid veins joining the internal jugular vein.
Dural venous sinuses – the venous drainage of the brain

When the dura lining the cranial cavity is intact, you can’t see the veins (dural venous sinuses) which give rise to the internal jugular vein. Compare the appearance inside a bony skull with that of a prosection of the cranial cavity with the dura intact. On the bony skull you can see grooves for the dural venous sinuses on the bone, while in the dural skull these channels are enclosed to form dural venous sinuses. Notice that there is a sinus in the region around the pituitary gland this is the cavernous sinus. These structures are properly part of the study of the central nervous system.

However it is clinically important that the dural venous sinuses (particularly the cavernous sinus) have valveless connections (emissary veins) with extra-cranial veins - these connections can carry infection into the meninges and brain. Look at a skull and find the following openings through which such emissary veins pass: superior orbital fissure, foramen ovale, parietal foramen and mastoid foramen (some of these connections are shown on the diagram below…)

Lymph nodes of the head and neck

Look at a superficial prosection of the head and neck and find some lymph nodes around the base of the head (submental, submandibular, parotid, posterior auricular) and also along the carotid sheath (jugulo-digastric and jugulo-omohyoid)
CARDIOVASCULAR MODULE
Physiology Laboratory

Friday, Week 6

The use of enzyme kinetics to detect markers of acute myocardial infarction
The Use of Enzyme Kinetics to Detect Markers of Acute Myocardial Infarction

Background

The principle of using serum marker proteins to diagnose tissue injury is based on (a) the markers being much more concentrated in tissue than in serum, and (b) certain proteins predominating in particular tissues. If these two conditions are met, tissue damage that releases proteins into the serum can be detected by an increase in the level of the protein in the serum, and the identity of the tissue damaged can be identified by the protein released. Some protein markers of acute myocardial infarction (AMI) are myoglobin, heart fatty acid binding protein, myosin light chain, cardiac troponin, creatine kinase (CK), lactate dehydrogenase (LD) and myosin heavy chain.

Isozymes are enzyme proteins that catalyse the same reaction. They have different structures and reaction kinetics, and are found in different proportions in different tissues. In the case of the two enzymes that can be used in infarction detection, CK and LD, the different structures arise because the enzymes are multisubunit proteins, made up of different combinations of either two (CK), or 5 (LD) available subunits. The different isozymes have unique isoelectric points and thus can be easily separated and identified using various forms of electrophoresis.

An example of benign tissue damage which can be detected using these isoenzymes is exercise like marathon running. In this case the levels of CK and LD increase in the serum due to muscle damage incurred during the run. The isozymes released are those found in skeletal muscle.

A more serious example is myocardial infarction which is a major cause of ill-health and death in Australia and in other developed countries. An ideal marker of myocardial injury (a) would be found in high concentrations in myocardium, (b) would not be found in other tissues, (c) would be released rapidly and completely after myocardial injury, (d) would be released in proportion to the extent of myocardial injury, and (e) would persist in plasma for long enough to be detected, but not long enough to interfere with the diagnosis of recurrent injury. The factors that determine these characteristics are size, cellular location, solubility, stability in plasma and detectability.

The principle of using serum enzymes to detect infarction was first used in 1954, and the enzyme used was aspartate transaminase. But since then more specific and more suitable enzymes such as LD and CK have become the markers of choice. Royal Perth Hospital has now gone one step further and is using one of the heart muscle proteins, troponin, that is part of the contractile machinery. This protein demonstrates tissue-specific isozymes and is apparently more definitive than either CK or LDH.

LD is a cytoplasmic enzyme found in most tissues in the body. The LD isozymes are numbered LD1-LD5. The isozymes can be separated using electrophoresis, all the isoenzymes are run as negative species and LD1 runs the fastest and LD5 the slowest. The heart contains predominantly LD1, but also LD2. Most of the...
LD in the body is found in the skeletal muscle, as the LD5 form. LD2, 3 and 4 are the dominant isoenzymes in the gallbladder, prostate and uterus. The ratio of LD1/LD2 in the serum is normally about 0.76. When cardiac necrosis occurs, more LD1 is released than LD2 and the ratio rises and can increase to >1.0. The rise is evident by 8-12 hours after the infarct, it peaks at 28-48 hours and is back to normal by day 10. However there are problems with this system as there are other possible causes of an abnormal ratio. For example, erythrocytes are an abundant source of LD1 and LD2 so intravascular or extravascular hemolysis can produce an elevated ratio, as can normal strenuous exercise, despite the fact that LD5 is the predominant form in skeletal muscle. And, sometimes the ratio does not change as a result of an AMI. The advantages and disadvantages of the various indicators of myocardial infarction are discussed in the following two recent articles, in case you are interested in reading more about it.


LD, as an indicator of infarction, is normally assayed using gel technology, and the isoenzymes of interest are 1 and 2 (see above). You will be using this type of method to assay CK in a later experiment in Biochemistry and Molecular Biology. In this experiment the aim is to show you how an activity assay, linked with isozyme-specific inhibitors, can be used to assess the relative proportions of different isoenzymes in an enzyme mix, such as the serum. We will not use LD 1 and 2, but LD 1 and 5, as the differential inhibition of these two isozymes is much more obvious. The principle is the same however as LD 1, but not 5 is found in the heart.
Objectives.
- To understand the principles of using serum proteins as specific tissue markers.
- To demonstrate how a kinetic enzyme assay can be used to distinguish an isozyme mix from a solution containing only one isozyme.
- To demonstrate that a myocardial infarction can be diagnosed on the basis of these principles.

The Experiment

Look up the reaction catalysed by lactate dehydrogenase, and make sure you understand its function in the cell. Also see p168 of your textbook for an explanation of how NADH can be used to measure enzyme activity.

You will be provided with 2 solutions, A is LD 5 only, B is a mixture of LD 1 and 5. You will assay these for LD activity under conditions of high and low pyruvate concentration. LD 1, but not LD 5, is inhibited by high pyruvate concentrations. This is due to the different affinities of the isoenzymes for pyruvate and NADH. NADH, once bound to LD 1, binds very tightly, and the presence of a high concentration of pyruvate can cause the formation of an NAD-pyruvate dead-end complex that is not released.
Method

1. Set up a microtiter plate as follows; **Note** You are using one column only.

<table>
<thead>
<tr>
<th>REAGENT</th>
<th>WELL</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1M Phosphate Buffer pH 7</td>
<td>2B</td>
</tr>
<tr>
<td></td>
<td>2C</td>
</tr>
<tr>
<td></td>
<td>2D</td>
</tr>
<tr>
<td></td>
<td>2E</td>
</tr>
<tr>
<td></td>
<td>2F</td>
</tr>
<tr>
<td></td>
<td>2G</td>
</tr>
<tr>
<td>1.5 mM NADH</td>
<td></td>
</tr>
<tr>
<td>Solution A (LD 5)</td>
<td></td>
</tr>
<tr>
<td>Solution B (Mix of LD 5 and LD 1)</td>
<td></td>
</tr>
</tbody>
</table>

2. When you have pipetted all the reagents into the wells, start the reaction by adding the appropriate concentration of pyruvate to wells 2D, 2E, 2F and 2G as indicated below. **Start your timer**

3. Then mix the wells with the multi-pronged mixer provided. Rinse the mixer in the beaker of water provided, after you have used it.

| 100 mM Pyruvate                  |       |
| 3.0 mM Pyruvate                  |       |

4. As soon as possible after you have added the pyruvate to the wells read the absorbance at 340nm.

5. Note the time of the first and subsequent readings, and within the next 30 min, read the absorbances 3 more times. Make these readings at least 5 min apart. Try 3, 10, 20 and 30 mins.

6. Plot graphs of Absorbance vs Time for each well.
If you want to plot them all on one graph in Excel, try following the steps below.

Go to Excel
1) Put your data in the spread sheet (e.g. first column - time, second column - absorbance of 2B, third column - absorbance of 2D and so on...)
2) Select any empty cell from other column
3) Click on the histogram icon in the tool bar
4) Choose XY scatter and click the picture with the dot only, then click “next”
5) The Data range box should be emptied, if no highlight everything and delete.
6) Click the button with a little red arrow (at the end of the data range box)
7) Outline all the filled columns
8) Click the button with a little red arrow again (a graph with six series in the legend box should be generated on top of the data range box, double check the x and y axis)
9) Click “next’ twice or until you see the “Chat Location” window
10) Select “As new sheet” and click finish
11) Right click on any point of a series
12) Add trendline, liner and then go to Options
13) Tick add equation and add R squared
14) OK
15) Repeat steps 12-15 until six equations and R squareds are obtained
16) You may need to adjust the position of the equations for easy visualisation
You may need to label the equations for identification

7. Determine the change in absorbance per minute for each well.

LAB REPORT

Answer the following questions.

1. If the LD solutions were serum samples from the same patient, and solution 2 was taken one week after solution 1, what could you conclude about the patient, and why? If your data is inexplicable, explain why this might be the case, and use another set of data that is more reasonable.
2. What were the controls for and why are they important?
3. What were you actually measuring in the plate reader, and why was it changing?
CARDIOVASCULAR MODULE
Anatomy Laboratory

Wednesday, Week 7

Blood vessels of the upper and lower limbs
Material used in this Laboratory:

Two sets of four tables

Station 1: Upper Limb – Arteries and angiograms on prosected and plastinated arms and hands
Station 2: Upper Limb – Veins in venograms and prosected and plastinated arms and hands
Station 3: Lower Limb - Arteries and angiograms on prosected and plastinated legs and feet
Station 4: Venous Drainage of the Lower Limb on prosected and plastinated legs and feet
NS 100  Cardiovascular system

**Laboratory class: Blood vessels of the upper and lower limbs**

Read and study these notes and use your textbook to become familiar with the contents BEFORE you come to the lab.

**Outcomes**

1. The student will be able to describe the vascular supply of the upper limb
2. The student will be able to explain the anastomoses of vessels which occur about the joints of the upper limb.
3. The student will be able to describe the vascular supply of the lower limb and the anastomoses found around the joints (periarticular anastomoses)
4. The student will be able to describe the pattern of venous drainage that involves superficial as well as deep veins in both the upper and lower limbs, and to appreciate the mechanisms that promote venous drainage, particularly in the lower limb.
5. The student will be able to recognise the importance of the axilla and inguinal regions for the lymphatic and venous drainage of the limbs.
6. The student will be able to take this opportunity to revise the material you have learned in this course before the lab test next week

Be prepared to identify the following structures both on a prosection and the surface markings where relevant.

Brachial artery, axillary artery, teres major, profunda brachii artery, radial artery, ulnar artery, flexor carpi radialis, anterior and posterior interosseus arteries, superficial palmar arch, common palmar digital arteries, proper palmar digital arteries, deep palmar arch, deep branch of the ulnar, venae commitantes, dorsal venous arch of the hand, cephalic vein, basilic vein, cubital fossa, median cubital vein, medial vein, profunda femoris artery, medial and lateral circumflex femoral arteries, perforating arteries, cruciate anastomoses, femoral artery, obturator artery, superior and inferior gluteal arteries, popliteal artery, genicular branches, anterior tibial artery, dorsalis pedis artery, arcuate arteries, 1st dorsal interosseus artery, posterior tibial artery, the peroneal artery, medial plantar artery, lateral plantar artery, dorsal venous arch of the foot, great saphenous vein, small saphenous vein, perforating veins, deep veins, saphenous opening, fascia lata, inguinal lymph nodes, femoral sheath.
Station 1 ARTERIES OF THE UPPER LIMB AND AXILLA

The **brachial artery** continues from the **axillary artery** beyond the **teres major** muscles at the lower border of the axilla. You can feel its pulse against the medial side of the humerus most of the way down the arm. The brachial artery travels with the medial nerve which can also be felt with the brachial pulse. The brachial artery gives off the **profunda brachii artery** which spirals around the posterior side of the humerus deep to the heads of triceps. It travels with the radial nerve which can be felt on the lateral side of the arm about halfway down. Find these vessels on a prosection of the arm. The profunda brachii usually gives off the nutrient artery to the humerus – look at some bones and find the nutrient foramen.

Just below the elbow (in the cubital fossa) the brachial artery divides into the **radial** and **ulnar arteries**. There are anastomoses around the elbow joint composed of branches of the brachial and profunda brachii arteries passing down and joining recurrent branches from the radial and ulnar arteries.

Use a prosection to follow the ulnar and radial arteries through the forearm. Most of the way they pass deep to the flexor muscles of the forearm but near the wrist the radial pulse can be felt lateral to the tendon of **flexor carpi radialis**. Try to feel the ulnar pulse lateral to the flexor carpi ulnaris tendon. Near its origin the ulnar artery gives off an **interosseus branch** which divides into posterior and anterior divisions which run deep through the posterior and anterior compartments of the forearm – look for these on deep prosections of the forearm.

The radial and ulnar arteries participate in a number of anastomoses (arterial arches) in the hand. The ulnar artery passes into the palm of the hand and arches to join a small branch of the radial artery in the plane between the palmar aponeurosis and the long tendons to the fingers. **This superficial palmar arch** gives off **common palmar digital arteries**, which divide in the webs of the fingers into **proper palmar digital arteries** to adjacent sides of the fingers.
The radial artery crosses the base of the thumb (anatomical snuff box and then enters the palm piercing the 1st dorsal interosseous muscle in the web between the thumb and forefinger). In the palm the radial artery becomes the deep palmar arch (anastomosing with a deep branch of the ulnar artery) which runs deep to the tendons to the fingers. Find these structures on deep dissections of the hand. There are two other arches: the anterior and posterior carpal arches – but these are small and not usually apparent on prosections.

Compare the arteriograms with the gross anatomy – can you see any pathologies? Why would you perform an arteriogram on a leg?

The axillary artery

Lateral to the edge of the 1st rib the subclavian artery becomes the axillary artery. It too has 3 parts partitioned by the pectoralis minor muscle. Each part has the following branches:

1st part Superior thoracic artery

2nd part Thoracoacromial artery that divides into clavicular, acromial, pectoral and humeral branches to the anterior part of the shoulder.

The lateral thoracic passes along the lower border of the pectoralis minor muscle and supplies the breast and pectoral region.

3rd part Subscapular artery is a large branch to the anterior surface of the scapular. It has a circumflex branch which passes around the lateral border of the scapula and anastomoses with the suprascapular artery. This anastomosis forms a major alternative pathway by which blood can enter the upper limb if the subclavian/axillary artery is compressed. Anterior and posterior circumflex humeral arteries (also from the 3rd part of the axillary artery) wrap around the neck of the humerus and anastomoses with each other.

Label the branches of the subclavian and axillary arteries shown in the diagram of the anastomoses around the shoulder.
Station 2 VENOUS DRAINAGE OF THE UPPER LIMB

1. Deep veins
The arteries are accompanied by venae commitantes, plexus of veins that surrounds the artery. These venae commitantes follow the radial, ulnar and brachial arteries. A true vein forms only in the axilla as the axillary vein.

2. Superficial veins
Superficial vein do not have accompanying arteries, they run in the superficial fascia (the fatty layer just below the skin). The main superficial veins begin as an arch on the dorsum of the hand (The dorsal venous arch of the hand). From the radial side of this arch the cephalic vein runs up the lateral side of the upper limb to the front of the shoulder where it passes between the deltoid and pectoralis muscles to join the axillary vein. From the medial or ulnar side of the dorsal venous arch the basilic vein continues up the medial side of the arm to just above the elbow where it pierces the deep fascia (the sheet of connective tissue that encloses the muscles and deeper structures of the limbs). It joins the venae committantes of the brachial vein. On the anterior aspect of the elbow (cubital fossa) there are connections between these superficial veins (eg. median cubital vein). Generally the valves in these veins direct blood towards the basilic vein.

How does this arrangement improve venous return from the limb.

The pattern of veins is highly variable and complex as there are many other veins as well (particularly one on the anterior aspect of the forearm called the median vein – it also joins the veins in the cubital fossa). Look at these veins on yourself and on your colleagues (use cuffs to restrict blood flow in the arm and enlarge the veins). Compare the pattern of veins and valves, identify the named vessels and see if the cephalic vein is reduced in size above the elbow.
Draw the pattern of veins in your antecubital fossa on the left and that of a colleague on the right, label the veins. Why do you think the bicipital aponeurosis is sometimes referred to as the Grace de Dieu ligament? (Grace of God ligament).
Station 3 LOWER LIMB - ARTERIES

EXTERNAL ILIAC ARTERY

The external iliac artery passes around the pelvic brim and leaves the abdomen by passing over the front of the hipbone behind the inguinal ligament (in the femoral sheath). Examine it on prosections and find the two branches it gives off to the abdominal wall before it leaves the abdomen:

Deep circumflex iliac passes laterally towards the iliac crest

Inferior epigastric artery passes up the anterior abdominal wall and enters the rectus sheath behind the rectus abdominus muscle where it anastomoses with the superior epigastric branch of the internal thoracic artery.

The lateral parts of the anterior abdominal wall are supplied by the last 6 posterior intercostal arteries which escape from their intercostal spaces and continue between the flat muscles of the abdominal wall (the 10th intercostal artery is directed towards the umbilicus).

Diagram of the posterior thigh

THE FEMORAL ARTERY

Is the continuation of the external iliac into the anterior thigh. It gives off a number of early branches to the body wall and perineum, but the important branches are to the hip and thigh:

Profunda femoris artery runs deep in the thigh and sends perforating branches to the back of the thigh.

Medial and lateral circumflex femoral arteries pass in either direction around the femur and anastomose at the back of the hip joint with each other and also the gluteal and the 1st perforating branch of the profunda femoris – this is the cruciate anastomosis of the hip joint.

Find these vessels on the prosections and also on the angiograms.

Indicate the CRUCIATE anastomoses
The profunda femoris artery usually gives off the nutrient artery to the femur – look at some bones and find the nutrient foramen, also look on the femoral neck for the many foraminae which enter the bone there.

Now follow the femoral artery down the front of the thigh to where it passes through the hiatus in adductor magnus to enter the popliteal fossa and become the popliteal artery. In the popliteal fossa it gives off a number of genicular branches which supply the knee joint and form the anastomosis around the knee.

Examine the angiograms showing the arterial supply of the lower limb and identify as many vessels as you can. In the space provided, draw a diagram of the branches of the popliteal artery and the arterial supply of the leg and foot (ie below the knee).

At the lower border of the popliteal fossa the popliteal artery divides into anterior and posterior tibial arteries. The anterior tibial artery passes to the front of the leg by passing above the interosseous membrane, then it runs deeply down the front of the leg. It emerges on to the front of the ankle where its pulse can be felt just lateral to the tibialis anterior tendon. It form an arch on the dorsum of the foot (dorsalis pedis and arcuate arteries) which then gives rise to 4 dorsal metatarsal arteries that head towards the webs of the toes. Look at prosections and find these vessels and also feel for their pulses (dorsalis pedis/1st dorsal interosseus).

The posterior tibial artery runs down the back of the leg deep to the gastrocnemius and soleus muscles. It heads for the sole of the foot by going behind the medial malleolus at the ankle. Feel for its pulse there. The posterior tibial artery gives off a branch in the leg – the peroneal artery which goes towards the lateral side of the ankle. Look on the prosections and find these vessels. The posterior tibial artery usually gives off the nutrient artery to the tibia – look at some bones and find the nutrient foramen.
In the foot the posterior tibial artery divides into the medial and lateral plantar arteries. The lateral plantar artery swings to the lateral side of the foot and then back towards the medial side near the metatarsal heads and it connects with the 1\textsuperscript{st} dorsal metatarsal (dorsalis pedis) artery by piercing the gap between 1\textsuperscript{st} and 2\textsuperscript{nd} metatarsals. Look at prosections of the foot and identify these main vessels.

Why is it clinically important to be able to locate the pulses of arteries in the foot?

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**Station 4 VENOUS DRAINAGE OF THE LOWER LIMB**

1. Deep veins

Venae commitantes follow the plantar, peroneal and tibial arteries. A true vein forms in the popliteal fossa (popliteal vein). The other important deep veins are found in the substance on the soleus and gastrocnemius muscles. These vessels are important for venous drainage of the whole limb as these two muscles are constantly active and continually compress and relax around these deep veins. This action (together with valves in these veins) pumps blood out of the leg and into thigh where the femoral vein is also surrounded by muscles and so this muscle pumping action continues. Look at the prosections to see the large veins inside the gastrocnemius and soleus muscles and the veins that leave these muscles and join the popliteal vein.

2. Superficial veins

As in the upper limb, the superficial veins begin at the dorsal venous arch of the foot. On the medial side of the arch the great saphenous vein begins and passes up the medial side of the limb to the inguinal region at the top of the thigh. The small saphenous vein comes from the lateral side of the dorsal venous arch, ascends the lateral side of the leg and empties into the popliteal vein behind the knee. Look at prosections and find these vessels. Their positions are important clinically because they are accessible to venepuncture. The great saphenous vein passes just in front of the medial malleolus and then just behind the medial side of the knee cap, and finally pierces the deep fascia at the saphenous opening a few centimetres below the midpoint of the inguinal ligament.
Especially on the leg the superficial veins have perforating branches that connect them to the veins inside the soleus and gastrocnemius muscles. These veins have valves that only allow blood to pass from superficial to deep. How does this arrangement assist venous return from the lower limb?

Why is the lower medial side of the leg a common site of venous ulcers in the elderly?

THE INGUINAL REGION AND AXILLA

Look at some prosections of the inguinal region at the top of the thigh. Find a very superficial dissection that shows some of the lymph nodes and superficial veins of the inguinal region. Notice how the great saphenous vein has to pass through the saphenous opening in the fascia lata, and that there are numerous inguinal lymph nodes in this region. They drain the lower part of the abdominal wall, the external genitalia, and the lower limb. Lymph then passes to deep nodes through the saphenous opening.

Look at a slightly deeper dissection of the inguinal region where the femoral triangle has been opened. Observe the femoral sheath, it is a sleeve of fascia extending from the lining of the abdomen that ensheaths the femoral artery vein and lymphatic vessels for a few centimetres into the thigh.

Find a superficial dissection of the axilla which shows some of the veins and lymph nodes found there. Lymph from the upper limb and superficial structures of the thorax drain to these nodes. Of course, in females this area is important because most of the lymph from the breast drains to the axilla. This topic will be dealt with in more detail during your study of the respiratory system.

Examine the angiograms of the upper and lower limb, revise the technique of angiography. Examine the differences between CAT scans and MRIs of the lower limb, compare both to conventional thin film X-rays. When would you use each technique?

Where would you feel for peripheral pulse in the lower limb. What would be the significance of weak or absent pulses? Why would diabetes be a complicating factor?
CARDIOVASCULAR MODULE

Physiology Laboratory

Friday, Week 7

Human blood groups
HUMAN BLOOD GROUPS

BACKGROUND
Blood groups are genetically determined antigenic characteristics of blood components. In this class you will be looking at some of the red blood cell groups. There are many blood group antigens on red blood cells but the most important ones, clinically, are those of the ABO and Rhesus systems. In this class you will determine the ABO and Rhesus(D) groups of two "unknown" blood samples and your own blood, collected by a finger-prick. You are provided with blood samples of known groups to use as controls. You will also consider the presence of naturally occurring antibodies and their significance in blood transfusions.

There are four ABO groups - A, B, O and AB. They are determined by the presence or absence of 2 antigens – the A and the B antigens – on the red blood cells. Cells that have only the A antigen are designated group A; cells that have only the B antigen are designated group B; cells that have both the A and B antigens are designated group AB; and cells that have neither the A nor the B antigen are designated group O.

The Rhesus antigens are numerous but the most important is the D antigen. Cells which possess this antigen are said to be Rhesus positive or more correctly Rhesus (D) positive, while cells without the antigen are said to be Rhesus negative or Rhesus (D) negative.

OBJECTIVES:

1. To learn how blood groups can be determined.

2. To examine the distribution of the ABO and Rhesus(D) groups in the class.

3. To understand the clinical implications of “naturally-occurring” blood group antibodies.

NOTE: Blood is potentially infectious, so handle it with care and clean up well. You should read the safety guidelines in the front of this manual on handling blood and follow the instructions of your demonstrator and the laboratory technicians.
PROCEDURES:

Collection of finger-prick samples

Make sure there is good blood flow to your hand by shaking or rubbing it or immersing it in warm water. Wipe your thumb or index finger with alcohol then jab it firmly with a blood lancet. The blood should be collected into the 5 ml vial containing citrate anticoagulant. The resulting suspension needs to be bright red in colour and cover the bottom of the vial to a depth of 2 to 3 mm to ensure you have enough cells to see the agglutination reactions.

Provided blood samples

You will be provided with 25% suspensions of human red cells of each ABO group. These cells will be of varying Rh(D) groups with at least one sample being Rh(D) positive and at least one being Rh(D) negative. Use these samples as controls for your blood group testing. You will also be provided with 2 “unknown” blood samples labelled “X” and “Y”. You will need to determine the ABO and Rh(D) groups of these samples. The cells may have settled while sitting on the bench and should be resuspended by gently upending the vials several times.

ABO Blood groups

We can detect the presence of the antigens on the red blood cells by testing the cells with antisera containing antibodies against the A or B antigens. When cells with the A antigen are mixed with anti-A antisera, the antibodies bind to the red cells and cause agglutination or clumping of the cells. Similarly cells with the B antigen will agglutinate when mixed with anti-B antisera.

1. Using a wax pencil, label your grouping trays with anti-A, anti-B and anti-A/B across the top and the type of cells to be tested down the side. The anti A/B contains both an anti-A antibody and an anti-B antibody and is used to check the reactions occurring with the other antisera. Test the four control samples (A, B, O and AB), the two unknown samples (“X” and “Y”) and your own samples.

2. Using the dropper bottles provided, add 1 drop of the appropriate antiserum to each well.

3. Using disposable pipettes, add 1 drop of a 25% suspension the appropriate cells to each well. (The provided samples have already been diluted to give 25% suspensions.)

4. Mix the contents of the wells with a toothpick, using a clean toothpick for each well.

5. Wait 2 minutes then gently rock the tray and look for agglutination or clumping of the cells. Record the agglutination patterns.

6. Record your agglutination results in the following table. Agglutination should be recorded as “+”; no agglutination as “−”.
<table>
<thead>
<tr>
<th>Blood Sample</th>
<th>Anti-A</th>
<th>Anti-B</th>
<th>AntiA/B</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>O</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>AB</td>
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<td></td>
</tr>
<tr>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Student 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Student 2</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

From this table determine the ABO blood groups of samples X and Y and the student samples.

X = ________________  Y = ________________

Student 1 = ___________  Student 2 = ______________

Rhesus(D) groups

The presence or absence of the D antigen can be determined using anti-D antiserum. Rhesus positive cells contain the D antigen and therefore they agglutinate with the Anti-D antiserum. Rhesus negative cells do not have the D antigen and so they do not agglutinate with the anti-D antiserum. You should test D+ve and D-ve control cells, the two unknown samples and your own samples.

1. Label microscope slides with the type of cells to be tested. Two samples can be tested on each slide.

2. Add 1 drop of anti-D and 1 drop of the cells to be tested.

3. Mix with a toothpick and place in a warm damp chamber for 5 minutes. (Place a paper towel soaked in warm water on the bottom of the plastic container. Place your slides in the containers, resting on the sticks provided. Do not put the glass slides directly onto the damp paper as they will be very difficult to remove. Place the lid on the container and leave for 5 minutes.)

4. Pick up each slide and rock it gently to look for agglutination. Record your results. (Note that these results are usually much weaker than the ABO agglutination reactions.)

5. Record the agglutination results in the table below, and determine the Rh(D) group of each sample.
Enter your ABO and Rh(D) groups on the class results spreadsheet. These results will be placed on WebCT after the classes. Summarise the results (using all the student data) on the results sheet provided in your lab book.

Compare the distribution of blood groups in the class with the distribution given for the Australian population.

Naturally-occurring antibodies

Most people have so called “naturally-occurring” antibodies in their plasma that react with the antigens of the ABO system. These antibodies develop during the first six months after birth without exposure to incompatible red blood cells. They are thought to develop in response to antigens on common bacteria that are similar in structure to the A and B antigens on human red blood cells. It is the presence of these antibodies in the plasma that causes ABO incompatible blood transfusions to result in immediate, life-threatening reactions.

You develop plasma antibodies (sometimes called agglutinins) against the antigen(s) that you don’t have on your own red blood cells.

Try to work out what antibodies will be in the plasma of individuals of each ABO blood type and complete the table below. To avoid confusion, antibodies should be written with the word “anti-” in front of them e.g. anti-A for an antibody that reacts with the A antigen. Check your answers with your demonstrator or textbook.
<table>
<thead>
<tr>
<th>Blood Group</th>
<th>Plasma Antibodies</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td></td>
</tr>
<tr>
<td>O</td>
<td></td>
</tr>
<tr>
<td>AB</td>
<td></td>
</tr>
</tbody>
</table>

There are no naturally-occurring antibodies in the Rhesus system but antibodies can develop if a Rh(D) negative person is exposed to Rh(D) positive cells. Such antibodies can cause delayed transfusion reactions on the first exposure to incompatible cells and can cause more serious problems in a subsequent transfusion or pregnancy. It is acceptable to give Rh(D) negative cells to a Rh(D) positive patient. Why?

**Before you leave the laboratory**, make sure that you have recorded your own blood groups on the class results spreadsheet and that you have cleaned up your work area.

**FOR YOUR REPORT**
Your report for this class is a web-based report which you must submit online before the advised deadline.

After completing the results tables on the previous pages, logon to the course website and answer the questions.
STUDENT BLOOD GROUP DATA

Sample size (n) =

<table>
<thead>
<tr>
<th>Blood Group</th>
<th>Number of students</th>
<th>% of students</th>
</tr>
</thead>
<tbody>
<tr>
<td>O</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AB</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rh(D)pos</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rh(D)neg</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Frequency of Blood Groups in Australia

<table>
<thead>
<tr>
<th>ABO groups</th>
<th>Rhesus(D) groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>O</td>
<td>D +ve</td>
</tr>
<tr>
<td></td>
<td>46.1 %</td>
</tr>
<tr>
<td>A</td>
<td>D -ve</td>
</tr>
<tr>
<td></td>
<td>39.0 %</td>
</tr>
<tr>
<td>B</td>
<td></td>
</tr>
<tr>
<td></td>
<td>11.4 %</td>
</tr>
<tr>
<td>AB</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3.5 %</td>
</tr>
</tbody>
</table>
CARDIOVASCULAR MODULE
Anatomy Laboratory

Wednesday, Week 8

Revision and test
CARDIOVASCULAR MODULE
Physiology Laboratory

Friday, Week 2

Red blood cell measurements and haemostasis
RED BLOOD CELL MEASUREMENTS AND HAEMOSTASIS

BACKGROUND

Red cell measurements. The number, size and composition of the erythrocytes and the haemoglobin concentration of the blood vary under different normal and abnormal conditions such as the age and sex of the subject and in response to hypoxia, haemorrhage and red cell destruction. Hence, much useful information about the functional state of the erythropoietic system can be obtained from an examination of the circulating red blood cells. In this class you will perform some simple red cell measurements on both a provided human blood sample and on your own blood samples obtained by a fingerprick.

The red cell measurements that you will be performing in this class are the haemoglobin concentration of the blood (Hb) which is measured in grams per litre of blood, and the packed cell volume (PCV) which is also known as the haematocrit and is expressed as a ratio. You will be given the red blood cell count (RBC), expressed as cells per litre of blood, for the provided human blood sample.

From these primary measurements you can determine other useful information about the red blood cells – the red cell indices: the mean corpuscular haemoglobin concentration (MCHC) which is expressed in g.l$^{-1}$, the mean corpuscular volume (MCV) which is expressed in fl (femtolitres i.e. $10^{-15}$ l) and the mean corpuscular haemoglobin (MCH) which is expresses in picograms i.e.$10^{-12}$ g). These parameters change characteristically in many pathological conditions of the blood. They are calculated as follows:

\[ \text{MCHC} = \frac{\text{Hb}}{\text{PCV}} \]
\[ \text{MCV} = \frac{\text{PCV}}{\text{RBC}} \]
\[ \text{MCH} = \frac{\text{Hb}}{\text{RBC}} \]

Haemostasis describes the events underlying blood clotting after blood vessel injury. Haemostasis involves vasoconstriction, activation of platelets and platelet plug formation, activation of the coagulation pathways leading to fibrin formation and the eventual removal of fibrin by fibrinolysis.

In this class you will be performing some simple screening tests of platelet function and the coagulation pathways. These simple tests will give you a good idea of a patient’s ability to form a blood clot and therefore prevent excessive bleeding. The tests will also give you some idea of the nature of the defect if a clotting abnormality is detected.
OBJECTIVES:

1. To learn how to measure the haemoglobin concentration of the blood (Hb) and the Packed Cell Volume (PCV or haematocrit).

2. To learn how the Hb and PCV can be used, along with the Red Cell Count, to determine other properties of the red blood cells – the Mean Corpuscular Haemoglobin Concentration (MCHC), the Mean Corpuscular Volume (MCV) and the Mean Corpuscular Haemoglobin (MCH).

3. To demonstrate the physiological variation in some of these measurements.

4. To learn how haemostatic function can be assessed.

5. To demonstrate the effects of aspirin on haemostasis.

PROCEDURE

Immediately upon entering this class, one student from each group should take 2 aspirin tablets (600 mg). The other student in each group will be the control student (no aspirin). Note the time of ingestion, as the Bleeding Time test needs to be performed 90 minutes after aspirin ingestion.

Please note: Students with any of the following conditions should not take aspirin: hepatic disease, kidney disease, uraemia, erosive gastritis, peptic ulcer, asthma, hypersensitivity to aspirin and other salicylates, G6PD deficiency, people taking anticoagulant therapy or who are pregnant. These students should act as the control subjects for this part of the class. Students with bleeding disorders should not participate as subjects for this class.

NOTE: Blood is potentially infectious so handle it with care and clean up well. You should read the safety guidelines in the front of this manual on handling blood and follow the instructions of your demonstrator and the laboratory technicians.
PART A: RED CELL MEASUREMENTS

On the provided human blood sample and on your own fingerprick samples you will perform one haemoglobin measurement and one PCV measurement.

Blood Haemoglobin Concentration

Haemoglobin concentration will be measured using a "Hemocue" Blood haemoglobin meter. Blood is drawn into the cuvette (by capillary action). Dry chemicals in the cuvette react with the blood to give the compound, azidemethemoglobin. The absorbance is measured at 570 nm and 880 nm and, after a delay of approximately 45 to 60 seconds, a value for haemoglobin is displayed in g.l⁻¹.

To Obtain a Fingerprick Sample

Make sure there is good blood flow to your hand by shaking or rubbing it. Wipe your thumb or index finger with alcohol then jab it firmly with a blood lancet If required apply light pressure until a drop appears, making sure the drop of blood is large enough to fill the cuvette completely. Introduce the cuvette tip into the middle of the drop of blood. The cuvette should rapidly fill in a continuous manner. It must be filled in one go as it cannot be topped up later. Carefully wipe off the excess blood from the outside of the cuvette. Air bubbles within the sample circle can produce erroneous readings. If present, you should discard the cuvette and try again.

Put the cuvette into the holder and gently push it into the stop point of the Hemocue. After approximately one minute the haemoglobin value will be displayed in the window.

Please consult the technical staff if you are in any way uncertain as to the correct operation of the instrument.

To fill the Hemocue cuvette from the provided vial of blood, tilt the vial and dip the tip of the cuvette into the blood. Hold it there until the cuvette is filled with blood, wipe the outside of the cuvette and read the result on the Hemocue as described above. Remember to resuspend the blood in the vial gently by slowly upending the vial several times before sampling.

Packed Cell Volume (also known as the Haematocrit)

This is measured by allowing blood to fill haematocrit tubes to approximately ¾ of their length, sealing the tubes and centrifuging in a microhaematocrit centrifuge. The haematocrit tube can be filled directly from your fingerprick or from the vial of provided blood (remember to resuspend the vial of provided blood by gently upending several times). Place the coloured tip of the haematocrit tube into the blood and carefully tilt so that blood runs up into the haematocrit tube. Stop filling the tube when the blood is about 1cm from the unmarked end. Take care to hold the tube horizontally and wipe off excess blood from the outside of the tube. Ensure that the unmarked end of the tube is both clean and dry (if not you will need to start again with a fresh tube). Seal this dry end of the tube by pushing it into a vertically held tray of putty several times until a firm plug seals the end of the tube. (Note that the putty does not seal after the lumen of the tube has been wet by blood, so seal the OTHER end to that used to draw up blood).
Place the tube in the centrifuge, sealed-end facing outwards and in contact with the rubber ring, and note the number of the well you have used. When most or all of the wells are occupied, screw on the flat plate which covers the samples, close the lid and set the timer for 5 minutes. After the centrifuge has stopped remove the haematocrit tubes and read the haematocrit using a microhaematocrit reader. Your demonstrator or technician can show you how to do this.

**Recording of results**

Haemoglobin concentration (g/L): \[\text{PCV} :\]

Provided sample:

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Student 1</td>
<td></td>
</tr>
<tr>
<td>Student 2</td>
<td></td>
</tr>
</tbody>
</table>

Using your results for the Hb and PCV of the provided sample and the given Red Cell Count (= \[\text{________ x 10}^{12}\text{ cells / L}\]), calculate the following values for the **provided sample** (Make sure you use the correct units).

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>MCV</td>
<td></td>
</tr>
<tr>
<td>MCH</td>
<td></td>
</tr>
<tr>
<td>MCHC</td>
<td></td>
</tr>
</tbody>
</table>

You can also calculate the MCHC (but not MCV or MCH) for your fingerprick samples

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Student 1 MCHC</td>
<td></td>
</tr>
<tr>
<td>Student 2 MCHC</td>
<td></td>
</tr>
</tbody>
</table>

**Make sure you record the results for your fingerprick samples (Hb, PCV and MCHC) on the class results spreadsheet.**
Analysis of class results

Using the whole class data from the spreadsheet on WebCT, calculate the male and female means and standard deviations and complete the following table:

<table>
<thead>
<tr>
<th></th>
<th>Hb concentration (g.L(^{-1}))</th>
<th>PCV</th>
<th>MCHC (g.L(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male mean</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male SD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female mean</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female SD</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Normal ranges (5\(^{th}\)-95\(^{th}\) percentile) for these measurements are shown below:

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC (x10(^{12})/l)</td>
<td>4.4 - 5.9</td>
<td>4.1 - 5.5</td>
</tr>
<tr>
<td>Hb (g/l)</td>
<td>130 - 170</td>
<td>110 - 150</td>
</tr>
<tr>
<td>PCV</td>
<td>0.40 - 0.53</td>
<td>0.36 - 0.49</td>
</tr>
<tr>
<td>MCV (fl)</td>
<td>82 - 98</td>
<td>81 - 98</td>
</tr>
<tr>
<td>MCH (pg)</td>
<td>26 - 31</td>
<td>25 - 31</td>
</tr>
<tr>
<td>MCHC (g/l)</td>
<td>310 - 350</td>
<td>310 - 350</td>
</tr>
</tbody>
</table>

PART B: TESTS OF HAEMOSTATIC FUNCTION

In this part of the class you will perform tests which assess different aspects of the haemostatic process - platelet plug formation, and the intrinsic and extrinsic coagulation pathways which lead to fibrin formation.

Whole Blood Coagulation Time

*Normal range (glass tubes): 5-11 minutes*

In this test coagulation is initiated by contact of the blood with the test-tube (intrinsic activation). The test will be performed as a class exercise on two student volunteers with your demonstrator performing the venepunctures. The effects of glass and plastic tubes will be examined.

1. 5 ml of blood will be collected by venepuncture and 1ml aliquots placed in each of 3 glass tubes and 1 plastic tube in a 37°C waterbath. Start the stopwatch.

2. Tilt the tubes about every 30 seconds to see if the blood has clotted. The end point is taken as that time when the tubes can be tilted past the horizontal position without blood flowing down the tube.

Record the times for all tubes on the class results spreadsheet.
Results: Whole Blood Coagulation Time (mins:seconds)

<table>
<thead>
<tr>
<th>Glass tube</th>
<th>Student 1</th>
<th>Student 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean for glass tubes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plastic tube</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Prothrombin time (Quick’s method)  
*Normal time: 12-14 seconds*

In this test, coagulation is initiated by the addition of Tissue Factor (the commercial name of the reagent we are using is Dade Innovan) to anticoagulated plasma samples. The effect of the anticoagulant is overcome by the presence of excess calcium in the reagent. Thus, in this test, activation of coagulation is via the extrinsic pathway. This test will be performed by each group of students on 2 standard plasma samples labelled as Plasma A and Plasma B (warfarin control). Each sample should be tested in duplicate.

The end point in this case is the first sign of clot formation or gelling. You may find this difficult to determine at first but, with experience, reproducible results can be obtained.

1. Check that your waterbath is at 37°C.

2. Place 10 drops of the tissue factor reagent (Dade Innovan) in a test-tube in the waterbath and wait a few minutes for it to warm up.

3. Add 2 drops of the plasma to be tested to a test-tube and place it in the waterbath to warm up.

4. Add 2 drops of the pre-warmed tissue factor reagent to the tube containing the plasma and start the stopwatch.

5. Tilt the tube and watch for gel formation. Record the time at which gel formation first appears.

6. Test each plasma twice and record the mean time for each sample.
Results: Prothrombin Time(s)

<table>
<thead>
<tr>
<th>PLASMA A</th>
<th>PLASMA B (Warfarin Control)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>1.</td>
</tr>
<tr>
<td>2.</td>
<td>2.</td>
</tr>
<tr>
<td>Mean:</td>
<td>Mean:</td>
</tr>
</tbody>
</table>

Bleeding time (Ivy's method)  
*Normal range 2-7 minutes*

As only small blood vessels are damaged in this procedure, blood loss will stop when the platelet plug forms, thus this test assesses platelet function. This test will be performed on each student by their partner. It should be performed at least 90min after the ingestion of aspirin by one student in each group.

1. Place a sphygmomanometer on the subject's upper arm and apply a pressure of 40 mmHg.

2. Swab the inside forearm with alcohol.

3. Make 3 quick, firm incisions in the forearm with a blood lancet and start the stopwatch. Take care to avoid superficial veins and scar tissue.

4. Gently blot blood away from the edges of the wounds every 15 seconds with the edge of the filter paper. Try to avoid wiping away the blood clot.

5. Record the time when blood ceases to flow from each wound.

Results: Bleeding Time (min:sec)

<table>
<thead>
<tr>
<th>Control Student</th>
<th>Aspirin Student</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td></td>
</tr>
</tbody>
</table>

Note: To calculate the mean values you will need to convert min:sec values into seconds.

Record the mean values for both Control and aspirin students on the class results spreadsheet.
Analysis of class results:

Using all the class data from the spreadsheet on WebCT, calculate the class mean for Bleeding Times of both the Control and Aspirin groups.

Class mean Bleeding Time for Control students = _____________________

Class mean Bleeding Time for Aspirin students = _____________________

Before leaving the laboratory, make sure you have filled in your results on the class results spreadsheet and have cleaned up your work area.

FOR YOUR REPORT

Your report for this class is a web-based report which you must submit online before the advised deadline.

After completing the data and calculations in your lab manual, logon to the course website and answer the questions.
RESPIRATION MODULE
Anatomy Laboratory

Wednesday, Week 9

The anatomy of breathing
Material used in this Laboratory:

Two sets of four tables

Table 1 Skeleton, prosections of intercostal muscles, ribs chest X rays, vertebrae
Table 2 Diaphragm, empty thoracic cavity on prosections, MRIs, CAT scans
Table 3 Prosections of the accessory muscles of respiration
Table 4 Larynx – larynx prosections and plastic models, video of swallowing
Read and study these notes and use your textbook to become familiar with the contents BEFORE you come to the lab.

General Aims and Objectives

6. The student will be able to describe the articulations and movements of the ribs and sternum.

7. The student will be able to outline the principal features of the diaphragm including its origin, insertion, actions and nerve supply, as well as the structures that pierce it, and its potential weaknesses.

8. The student will be able to list the accessory muscles of respiration and describe how they contribute to increasing thoracic volume.

9. The student will be able to identify and name the pleural cavity; its parts, function and its relationship to the rib cage. Also to develop an understanding of the sensory nerve supply of the pleura.
Station 1: THE CHEST WALL

1. Examine an articulated thoracic skeleton:

How many pairs of ribs form the thoracic cage? _______________________________

How many pairs of ribs articulate directly with the sternum through their own costal cartilage? ________________________________ What are these ribs called? ________________________________

How many pairs of ribs articulate with the costal cartilage of the rib above? ________________

What are these ribs called? ________________________________

How many pairs of ribs end without articulating with anything? ________________________________

Draw a diagram of the anterior aspect of the sternum and label its component parts:

- Manubrium
- Sternal angle
- Body
- Xiphoid process

Add the costal cartilages of the first 7 ribs to the diagram of the sternum.

2. Examine loose ribs and draw a diagram of a 3rd to 6th rib showing:

- Head
- Neck
- Tubercle
- Angle
- Body
- Costal groove
4. Examine a mid-thoracic vertebra and a rib. Using the skeleton and your text book, try to work out how each rib articulates with the vertebral column. Draw a diagram of the articulation below indicating the costovertebral and costotransverse joints (suggest superior view):

How does the rib move in the typical situation drawn above (draw a line on the diagram to show the axis of movement)? ____________________________

How does the articulation/movement of the ribs differ for
a) False ribs? (8 to 10) ____________________________

b) Floating ribs? (11 & 12) ____________________________

Make brief notes on the mechanisms by which the volume of the thoracic cavity is increased during filling of the lungs.

Sternum ____________________________

Upper ribs ____________________________

Middle ribs ____________________________

Lower ribs ____________________________

Now examine the chest wall of a cadaveric specimen:

Note the arrangement of intercostal muscles.

6. Which structures run immediately below each rib (partly hidden in the costal groove)?

________________________________________________________________________

________________________________________________________________________
Station 2. THE DIAPHRAGM

Examine the specimen showing the diaphragm (there is also a good bottle with the diaphragm available in the resource centre (G04))

Use the diagram provided (It’s a view from the abdominal aspect) to label the following features of the diaphragm:

- Xiphoid slips
- Costal margin
- Central tendon
- Opening for the IVC
- Oesophageal hiatus
- Aortic opening
- Left and right crus
- Median arcuate ligament
- Medial arcuate ligament
- Lateral arcuate ligament
- 12\textsuperscript{th} rib

Note that the IVC penetrates the central tendon but the Oesophagus is surrounded by muscle as it pierces the diaphragm. How might this anatomical difference effect the movement of substances in the IVC and oesophagus?

At what vertebral levels do the three main structures pierce the diaphragm?

- IVC
- Oesophagus
- Aorta

What other structures penetrate the diaphragm?

- 
- 
- 
- 
- 
- 
- 

4+
What structure is firmly attached to the upper surface of the central tendon

How does the central tendon move when the diaphragm contracts?

Which part(s) of the diaphragm muscle are most important/strongest?

What is the nerve supply of the diaphragm?
Motor? ____________________________
Sensory- to middle part? ____________________________
Sensory- to periphery? ____________________________

Look at the MRIs and CAT scans provided – can you recognize the diaphragm? Note its relationships to surrounding structures. Can you see why liver pain may be referred to the shoulder region?
Station 3. ACCESSORY MUSCLES OF RESPIRATION

Look at the prosections showing the accessory muscles of respiration. Identify each of the muscles (or muscle groups) in the list below and for each muscle say how it might contribute to breathing and whether it would help in inspiration or expiration.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Action</th>
<th>Stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pectoralis major and minor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serratus anterior</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

What else must be done to make these effective respiratory muscles?

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Action</th>
<th>Stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sternocleidomastoid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scalene muscles</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Erector spinae group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serratus posterior superior</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serratus posterior inferior</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quadratus lumborum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdominal wall muscles</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pelvic floor muscles</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Look at the diagram of the abdominopelvic cavity and the three muscles that enclose it (Diaphragm, Pelvic floor, and the Anterior abdominal wall). Consider how the actions of those muscles can effect bodily functions like breathing, coughing, defaecating, urinating, vomiting etc.
Station 4. TRACHEA AND EXTERNAL LARYNX

Relations of the trachea and bronchi

Where does the trachea begin?  Vertebral level ________________
   Bottom of the…_____________________
   Named cartilage ____________________

Where does the trachea end?  Vertebral level ________________
   Landmark on anterior chest __________

What structure lies behind the trachea through its course ________________________

What structures lie in front of the lower part of the trachea (ie. in the superior mediastinum) ____________________________

What organ is closely applied to the (sides of the) upper part of the trachea? ____________________________

What other structures lie lateral to the trachea in the neck? ____________________________

What nerves ascend along the posterolateral edges of the trachea (and terminate in the larynx) ____________________________

What is a tracheostomy? ____________________________

What structures lie in front of the trachea in the neck, that would have to be avoided in a tracheostomy? ____________________________

Examine the hemisected wet specimens and the models of the larynx and identify the following cartilages and bones:

Hyoid bone – horseshoe shape + greater and lesser horns

Epiglottis – leaf shaped elastic cartilage

Thyroid cartilage – left and right laminae, anterior prominence, superior and inferior horns, open posteriorly

Cricoid cartilage - Anterior ring and posterior lamina

Cricothyroid membrane – is below the level of the vocal cords

 Arytenoid cartilage – Apex, vocal process, muscular process
Note that:

1. The arytenoid cartilages rest on top of the cricoid lamina
2. The arytenoid cartilages give (posterior) attachment to the vocal cords
3. Movements of the arytenoids open and close the vocal cords
4. The anterior attachment of the vocal cords (ligaments) is inside of the thyroid prominence
5. There is a joint between the cricoid and thyroid cartilages that allows the two cartilages to move and stretch the vocal cords

IF available – look at the video of swallowing.
RESPIRATION MODULE
Physiology Laboratory
Friday, Week 9

Spirometry
SPIROMETRY

Background

The general aim of this class is to measure lung function in humans. The tests of pulmonary function to be performed in the class are:

1. **Lung Spirometry** with measurements of:
   
   i) tidal volume $V_t$ (L)
   
   ii) respiratory frequency $f$ (breaths/min)
   
   iii) ventilation $V_e$ (L/min)
   
   iv) forced vital capacity FVC (L)
   
   v) forced expiratory volume in 1 second FEV$_1$ (L and as a % FVC)
   
   vi) peak expiratory flow rate PEF(R) (L/min or L/sec).

   These are recorded using a pneumotachograph (sometimes simply called a flow head) attached to an amplifier and the Powerlab. This device gives the flow signal which is then integrated by the Powerlab to give the volume signal.

2. **Alveolar O$_2$ and CO$_2$:** CO$_2$ and O$_2$ %’s are recorded, then converted to partial pressures, during normal breathing, hyperventilation and breathholding.

3. Measure the **height** and **weight** of each subject.

Objectives

- A knowledge of ‘normal’ lung volumes, and variability in a population.

- A knowledge of partial pressures O$_2$ and CO$_2$ in respiratory system.

- Understanding the main factors which determine maximum expiratory flows (as assessed here by PEFR and FEV$_1$).

- Understanding the interpretation of simple tests used in distinguishing between obstructive and restrictive patterns of lung disease.

Experiment

*Work in pairs. Each student should obtain his/her own individual data. Rotate between the apparatus for spirometry and for recording gases.*
1. Lung Volumes/Spirometry

Summary
Take about 5 normal breaths through the flowhead, then a maximum inspiration followed by a maximum and forced expiration. Ensure complete emptying of lungs. Finish with one last tidal breath.

Do this twice, use readings with greatest PEF for your data.
Get printouts of data, spirogram and flow-volume loop.
Fill in your own data sheet.
Record predicted values from the charts on the wall

To begin the experiment follow the instructions below.

The file will be open and ready to go. You will see something like this.

ON NO ACCOUNT SHOULD YOU ATTEMPT TO ADJUST THE SETTINGS OR CLOSE DOWN THE FILES (unless directed). IF YOU DO YOU WILL LOSE THE PROGRAM AND WILL HAVE TO WAIT FOR SOMEBODY TO FIX THE PROBLEM.
To Operate

1. Click on the **start** button (bottom right hand side of screen) and observe the lower trace.
2. Place your blue filter mouthpiece into the end of the flowhead and place your nose clip on.
3. Whilst looking away from the screen, breath normally, in and out of the flowhead, for at least 5 breaths.
4. Then breath in as deeply as possible and exhale as fast and hard as you can until you have emptied your lungs. Finish with one last tidal breath. You can then remove the flowhead from your mouth and **stop recording**, by again clicking the "**start/stop**" button.
5. To obtain the results from your trace you need to highlight the area of the trace by clicking and dragging over all the breaths **on the lower trace**, right up to the final tidal breath (see below).

6. Go to the menu "**spirometry**" and highlight "**report**", the values will then be displayed. Check that they are reasonable, you will need to repeat the experiment and select the trace with the best PEF as the one to record your data from. When you are satisfied your trace is reasonable you can obtain a hard copy of this data, plus a plot of the flow-volume relationship. To do this go to the "**file**" menu and select "**print spirometry report**". You will need to click on "**ok**" at the next prompt. Print the Flow-Volume plot after selecting "**Flow-Volume**" plot from the "**Spirometry**" menu.

7. Now we wish to look at and print the spirometry trace. Go to the "**Spirometry**" menu and select "**Data Window**". To obtain a hard copy, such as the one printed below, go to the file menu and select "Print Spirometry Data". (It is best to set printer to print in "Landscape" mode). Again you will need to click on the print icon to activate the printer. Then click "**ok**" at the next prompt.
When you have collected all your data the next subject can continue using the same file. Just use **start** and **stop** buttons.

2. Partial Pressures of CO$_2$ and O$_2$

   A. Insert your disposable filter into the Haldane tube and apply the noseclip. Notice that a fine cannula leads to the gas metre which continuously draws air from the mouthpiece region. Channel 1 on the Powerlab shows the % O$_2$ and channel 2, % CO$_2$, %CO$_2$ and %O$_2$ can be measured by placing the cursor over the required section of trace on the Powerlab, and clicking the mouse once.

   The equipment will be set up and ready to start, click on the **start/stop** button on the bottom right hand corner of the computer screen to begin recording.

   B. (1) Normal breathing. Place the tube into your mouth and breath normally for 3 or 4 breaths. After the last normal exhalation, continue to breath out fully until no further air can be exhaled. Leave your mouth in place for 10-15 seconds after the exhalation so that the DATEX can sample your alveolar air. Once you have finished click on the "**start/stop**" button to stop the recording. From the trace on the Powerlab, record the peak CO$_2$% and the lowest O$_2$% when a steady level is achieved (see example trace below), this is done by placing the curser on that portion of the trace you wish to record values and clicking - enter these values on the Subject Results Sheet. [NB. Do not breathe more than 3 or 4 times through the tube - WHY?]
(2) Notice the swings in CO₂ during an inspiration and an expiration. Record the O₂ and CO₂ of fresh air. The trace should look something like the one below.

Before moving to the next experiment ensure you have obtained accurate data from your recording. When you are ready to proceed click on the start/stop button.

C. (1) After hyperventilation - (deep breathing). Rest for a minute, breathing normally. Hyperventilate for 15 seconds (do not use the Haldane tube) and then exhale fully into the Haldane tube. Record % CO₂ and O₂.

(2) After breathholding - Rest for a minute or two, breathing normally. Hold your breath at a comfortable lung volume (mid tidal) for as long as possible then again exhale fully into the Haldane tube. Record % CO₂ and O₂.
Assessment

There is an on-line assessment for this lab. Instructions will be provided in the lab.

1. Examine and understand the spirometer trace showing the tidal volume and forced vital capacity and the flow-volume trace. Compare your values with normal predicted values.

2. You tabulated your % CO₂ and O₂ values obtained during normal breathing, hyperventilation and breathholding. You will now need to convert %’s to partial pressures (the barometric pressure on the day, will be provided). Assume that the gas measured for % O₂ and CO₂ was dry (i.e. that the values recorded are fractional concentrations in dry gas).

Subject: __________________________________________

Age: _______ Height: _____(cm) Weight: ________ (Kg)

1. Spirometry

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Reading</th>
<th>Predicted</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tidal volume Vₜ (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>V’ E (1/min)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiratory frequency (b/min)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Forced vital capacity (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FEV₁ (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PEFR (1/min)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FEV₁/FVC (%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Complete the following table for your measurements of PA_{CO₂} and PA_{O₂} during normal breathing, hyperventilation and breathholding.

<table>
<thead>
<tr>
<th>Measured alveolar gases</th>
<th>CO₂</th>
<th>O₂</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>mmHg</td>
</tr>
<tr>
<td>Normal Breathing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperventilating</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breathholding</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
RESPIRATION MODULE
Anatomy Laboratory

Wednesday, Week 10

Histology of the respiratory system
Material used in this Laboratory:

Histology Lab microscopes

Permission to log on to histology system
Read and study these notes and use your textbook to become familiar with the contents BEFORE you come to the lab. This laboratory will be held in the histology lab opposite the DR.

Outcomes

1. The student will be able to describe the basic microscopic structure of the respiratory system, in terms of the characteristic mucosa, submucosa, muscularis and cartilage components.
2. The student will be able to identify parts of the respiratory system and distinguish them from other non-respiratory structures.
3. The student will be able to describe the structure of the lungs and bronchial tree.
4. The student will be able to identify the main parts of the upper respiratory tract, especially, larynx, epiglottis, nasal cavity and sinuses.
5. The student will be able to recognize the structural relationships between the respiratory and lymphatic systems.

TRACHEA

Examine the slide of the trachea under low power and identify the layers:

1. Mucosa consisting of respiratory epithelium and the lamina propria
2. Submucosa
3. Cartilage and muscle which in the trachea consists of a horseshoe shaped cartilage completed posteriorly by smooth muscle (trachealis muscle)

Examine the sections of the trachea under higher power and identify:

1. The features of respiratory epithelium

What is the full name of respiratory epithelium _____________________________

Name two kinds of cells in the epithelium and indicate how each functions to clean and protect the lungs.

______________________________________________________________

2. What structures can you see in the lamina propria?

______________________________________________________________

Can you relate this to the function of the respiratory passages

______________________________________________________________
3. What is seen in the submucosa of the trachea

How does this assist in the function of the trachea?

4. What kind of cartilage is found in the walls of the trachea

What is the function the cartilage in the respiratory system?

5. Can you see an adventitia outside the cartilage and muscle zone? What other structures are nearby?

**BRONCHUS**

Look at a section of a bronchus and identify the same features that you saw in the trachea. What differences can you see, and how might they relate to the function of the bronchus?

- Mucosa
- Submucosa
- Cartilage

Can you see a blood vessel adjacent to the bronchus? Would this be an artery or vein?

Comment on the differences you notice between it and a similar sized vessel in the systemic circulation.

**BRONCHIOLES**

Look at a part of the lung that has a bronchiole. What are the main differences between a bronchiole and a bronchus?
Comment on the functional significance of this

ALVEOLAE
Find a part of the lung that shows a terminal bronchiole, a respiratory bronchiole and an alveolar duct surrounded by alveolar sacs.
What is the difference between a terminal bronchiole and a respiratory bronchiole or an alveolar duct?

Look at the walls of an alveolus using high power
Name the cell types comprising the lung "alveolus"

What sort of epithelium lines the alveolar sacs?
What else is found in the walls that separate one alveolus from another?

Look at an electron micrograph of an alveolar wall to identify the structures involved in gaseous exchange.

NASAL CAVITY AND SINUSES
Examine a slide of the nasal cavity or paranasal sinuses. Comment on the similarities and differences between this organ and the trachea or bronchus.
Epithelium
Lamina propria
Submucosa
Cartilage/muscle

EPIGLOTTIS
Look at the slide of the epiglottis.
What sort of cartilage forms the core of the epiglottis?
Look carefully at the epithelium on each side of the epiglottis. You should notice that it is different on the two sides. For each side indicate the epithelium and state whether the side faces the tongue...
or the larynx

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Epithelium</th>
</tr>
</thead>
</table>

Why isn’t the epiglottis coated with respiratory epithelium on both sides?
TONSILS
Examine a slide of the tonsil. Comment on the similarities between this section of a tonsil and a lymph node or spleen.

What kind or epithelium coats the surface of this tonsil?
What is unique (structurally) about this epithelium?

With that sort of epithelium, do you think this tonsil is: palatine or pharyngeal (adenoid)?

What sort of epithelium do you think the other tonsil will have? Why?

What is the functional importance of "tonsillar crypts"?
RESPIRATION MODULE
Anatomy Laboratory

Wednesday, Week 11

The lungs
Material used in this Laboratory:

Two sets of four tables

STATION 1: The Hilum of the Lung – Laryngoscopes -Lungs in Buckets, X rays, CT, MRI and Skeletons

STATION 2: The Lungs – External Features - Lungs in Buckets –FRESH LUNGS

STATION 3: Pleural Cavities, The Lungs – Internal Features - Lung Models - Radiographs

STATION 4: The lungs – surface anatomy – drawings, body projections – data projector
Read and study these notes and use your textbook to become familiar with the contents BEFORE you come to the lab.

Outcomes

1. The student will be able to describe the parts of the air conduction system: larynx, trachea, bronchi, their functions structure and locations
2. The student will be able to describe the relations of each lung, especially those features that leave post-mortem impressions on the lung surface.
3. The student will be able to identify the structures seen in the hilum of each lung and to relate those to structures found in the mediastinum. Also to develop and understanding of the pattern of lymph drainage from the lung.
4. The student will be able to outline the branching pattern of each bronchus and its relationship to the lobes and bronchopulmonary segments of the lung. Also to understand how the branches of the pulmonary arteries and tributaries of the pulmonary veins relate to the bronchi.
5. The student will be able to draw the surface markings of the lungs and the pleura on the chest of a fellow student (surface anatomy).
Station 1. THE HILUM OF THE LUNG

Can you identify the tubular structures found in the hilum? __________________________
How? ____________________________________________________________________________
__________________________________________________________________________________

In the systemic circulation what is the obvious difference between arteries and veins that can be seen with the naked eye? __________________________
__________________________________________________________________________________

Is this difference so apparent in the pulmonary circulation? ________________
Explain __________________________________________________________________________
__________________________________________________________________________________

What are the solid black structures seen in the lungs hilum? __________
What is the name of this group ___________________________________________________________________
There is another group at the bifurcation of the trachea called __________

Consider the differences in the relative positions of structures in the hilum of the right and left lungs (look at the figure on the previous page and also the figure on the next page)
Can you see any Bronchial arteries? What are bronchial arteries?

__________________________________________________________________________________

A laryngoscope and a mirror have been provided for you to experiment with.

Look at the X-rays, CAT and MRIs provided – compare these with the lungs and skeletons.
Station 2. THE LUNGS – EXTERNAL FEATURES

Examine the right and left lungs found in buckets and also those still in the thoracic cavity. Consider the structures that lie in contact with the lungs and locate the following areas:

**On right and left lungs:**
- Diaphragmatic surface and the costodiaphragmatic border
- Costal surface with the costomediastinal border and vertebral border
- 1st rib and apex
- Mediastinal surface with cardiac impressions (which heart chambers)
- Hilum and Pulmonary ligament

Of course the right lung has 3 lobes and the left lung has 2 lobes, but how else might you determine whether an isolated lung is from the left or right side?

**On the right lung**
- SVC and azygos vein
- Oesophagus
- IVC
- Ascending Aorta

**On the left lung**
- Aortic arch
- Descending aorta
- Oesophagus
- Left subclavian artery

Are these grooves and other impressions present in life? ______________

Look at the surface of the lungs and you will see polygonal fields about 15mm across, these are bases of **secondary lobules supplied by a bronchiole of 1mm diameter**. The black markings that outline these fields are inhaled pigments contained in the lymphatic vessels at the boundaries of the lobules. If you look more closely you can see finer lines surrounding the **lung units each served by a respiratory bronchiole**.
Fresh lungs are set up in a side room (as we cannot mix animal and human material). Note the spongy texture of the unfixed lung. The demonstrator will show how the lung can be **gently** inflated – note how the elastic recoil causes the lung to collapse without any muscular action. This is what happens when the patient has a pneumothorax.

What is a pneumothorax? What might cause this?

What is a haemothorax? What might cause this?

Just for interest:-
A chyllothorax is when the pleural cavity fills with lymph What might cause this?

Hydrothorax (fluid in the pleural cavity) can be caused by cancer of the liver – can you guess why this can happen?
Station 3: THE LUNGS – INTERNAL FEATURES, PLEURAL CAVITIES

Examine the partly dissected lungs, the models of the lungs and bronchial tree, and the radiographs provided.

How many lobes do the lungs have? Right ________ and Left ________

What are the lobes called? Right ____________________________ Left ____________________________

What kind of bronchus supplies each lobe? ____________________________

How many Bronchopulmonary segments are there in each lung and lobe

Right ____________________________ Left ____________________________

What kind of bronchus supplies each bronchopulmonary segment? ____________

Notice that each branch of the bronchus is accompanied by a branch of the pulmonary artery. These pass into the centre of each bronchopulmonary segment. They have a constant relation to each other: The artery is:

Superior ________ to horizontal bronchi

__________ to ascending bronchi

__________ to descending bronchi

Also be aware (see figure two pages back) that the tributaries of the pulmonary veins lie in the boundaries of bronchopulmonary segments.

Consider the differences in the left and right bronchi:

Which bronchus is larger ________

Why ____________________________

Which bronchus is more vertical _____

Why ____________________________

Into which bronchus is an inhaled object more likely to pass? ____________________________
Pleural cavities

Explore the pleural cavities, using a specimen where the thorax has been opened and the thoracic viscera, are in situ.
Where is the visceral pleura?

What is parietal pleura?
Identify the following parts of the parietal pleura and give the nerve supply of each:
Costal pleura?
Mediastinal pleura?
Diaphragmatic pleura?
Cervical pleura (cupola)?

What is the sensory nerve supply of the visceral pleura?
What is the pulmonary ligament?

The lung completely occupies the pleural space inside the thorax except in places where the cavity is very narrow. These places are called pleural recesses. Use your gloved hand to explore the Costodiaphragmatic recesses (left and right) in the angle between the diaphragm and the rib cage. The costomedial recess is only found on the left hand side in front of the heart.
Station 4. SURFACE ANATOMY

In preparation for the tutorial on the surface anatomy of the lungs and pleura you should consider why it is important for a medic understand the surface projections (ie surface anatomy) of the pleura, lungs, diaphragm. How do the numbers 2, 4, 6, 8, and 10 help you remember?

Draw these features on each other using the crayons provided.
Surface markings of lungs and parietal pleura on the anterior thoracic wall.

Rear View
Images from Snell – Clinical Anatomy by Systems Lippincott and Williams
RESPIRATION MODULE
Physiology Laboratory

Friday, Week 11

Respiratory and metabolic acidosis
Respiratory and Metabolic Acidosis

Background
In the second year of your course, in the Renal Normal System, you will be given a series of lectures and a tutorial on acid / base balance. These lectures are based on a book called "Acid-Base and Electrolyte Balance" by R.J. Mead. This book will be available for purchase next year, but it would be a good idea to have a look at it this year as it will help you to understand this experiment. There are 7 copies in both the Biological Sciences and Medical Libraries.

The experiment today gives you a practical demonstration of two common perturbations of the acid / base status of human blood/extracellular fluid. These phenomena will be addressed more fully in the second year of your course.

Most of the metabolic processes in the body result in acid production, and there are some situations, e.g. vomiting that can result in excretion of acid. But despite these fluctuations, under most circumstances, the pH of the extracellular fluid of a human is tightly regulated between 7.35 and 7.43. This is equivalent to a [H+] of about 40 nM, with a range from about 25 to 100 nM. pH regulation is important because so many processes in the cell are affected by pH.

There are six buffering systems in the body, which include the bicarbonate-carbonic acid system, two protein-based systems, a phosphate system and two systems based on ion exchange.

The system we are concerned with today is the bicarbonate-carbonic acid system, which operates in the extracellular fluid. This system is actually quite complicated, and it is difficult to see how it is so effective when the buffering species (carbonic acid) has a pKa of 6.1, far below the pH at which the extracellular fluid is regulated. These aspects will be discussed in more detail next year.

For the purposes of this experiment we can simply describe this system as the following equation.

(1) dissolved CO₂ + H₂O  ⇄ H₂CO₃ (carbonic acid) ⇄ H⁺ + HCO₃⁻.

As only a very small proportion of the dissolved CO₂ is in the form of carbonic acid, equation (1) can be simplified to:

(2) dissolved CO₂ + H₂O  ⇄ H⁺ + HCO₃⁻.

and the Henderson and Hasselback equation becomes:

(3) pH = 6.1 + log [HCO₃⁻]/[dissolved CO₂]
CO\(_2\) is constantly being produced, so is regulated at 40 mmHg in the blood, high enough to facilitate efficient and continual transfer into the air, of the CO\(_2\) that is produced. In order to maintain CO\(_2\) at 40 mm Hg in the blood (which translates to 1.2 mM dissolved CO\(_2\)), the [HCO\(_3^-\)] is kept at about 24 mM. If these numbers are put into equation (3), you can see that the pH of the extracellular fluid = 7.4.

So you can see that the implications of this system are that the pH of the extracellular fluid is dependent on not only \([H^+]\), but on \([HCO_3^-]\) and [dissolved CO\(_2\)] as well. These interactions combine to produce two common perturbations of the pH of extracellular fluid in humans, termed metabolic and respiratory acidosis.

**Respiratory Acidosis**
The simplest type of respiratory acidosis (acute) can be caused by a sudden pulmonary infection or a bronchial obstruction. Both of these situations result in decreased alveolar ventilation and thus decreased CO\(_2\) exchange. The arterial pCO\(_2\) rises and (see equations 2 and 3), [HCO\(_3^-\)] rises and pH falls. This situation is easily and simply treated by treating the infection, or by clearing the obstruction, both of which restore the arterial pCO\(_2\). However, respiratory acidosis can be accompanied by a hypoxic metabolic acidosis, or can result in compensation by a metabolic alkalosis. The latter especially can be difficult to diagnose as the pH is normal, but the pCO\(_2\) is high, which can also be the result of metabolic alkalosis with respiratory compensation.

**Metabolic Acidosis**
This is the overproduction of acidic metabolites and can occur as a result of:
1. Ketoacidosis as a consequence of diabetes or starvation, or fever (which also results in reduced food intake).
2. Violent exercise or convulsions, or some form of hypoxia.

Look at equation (2). If the pCO\(_2\) remains constant, and [H\(^+\)] rises, the [HCO\(_3^-\)] will decrease, and the pH (equation 3), will fall. This is called uncompensated (acute) metabolic acidosis. This decrease in pH can be avoided by decreasing the pCO\(_2\), and the situation is then termed compensated (chronic) metabolic acidosis. It can also be treated by administering HCO\(_3^-\) (see equation 2), but bicarbonate infusion can be very dangerous (see Acid-Base and Electrolyte Balance, by RJ Mead, Chapter 5).

**Objectives**
- To understand the principles of buffering by the bicarbonate-carbonic acid system.
- To use a simple system to demonstrate a respiratory and a metabolic acidosis.
- To manipulate the system to demonstrate how a respiratory acidosis can be corrected by changing the pCO\(_2\), and how a metabolic acidosis is refractory to pCO\(_2\) and must be corrected by other means.
The Experiment

Method
1. You are provided with a 15 ml volume of 5 mM phosphate buffer at pH 7, containing about 1U/ml of carbonic anhydrase (which catalyses equation (1)). The enzyme simply accelerates all the changes, but does not alter the equilibrium of the reaction.

2. Set up your pH probe inside the tube containing the airstone, add the 15 ml of buffer and leave the probe in the buffer for the rest of the experiment.

3. Bubble the solution with air. This will create our "normal blood", even though air has a pCO₂ of only 0.22 mm Hg. It should take 3-5 minutes for the pH to stabilise. Record the pH.

4. The "patient" now gets a bronchial obstruction. Simulate this by turning on the gas cylinder and bubbling the CO₂ gas mix (5% CO₂ in air = 38 mm Hg) through your blood. Record the change in pH every 15 seconds.

5. Once the pH has stabilised, remove the flow of CO₂. You now have a respiratory acidosis. The concentrations of dissolved CO₂, HCO₃⁻ and H⁺ have increased and the pH has decreased.

6. You will now simulate the removal of the obstruction, increase bronchial ventilation, lower dissolved CO₂ and correct the respiratory acidosis. Simulate this by bubbling air through your blood. Record the change in pH every minute. Keep bubbling until the pH has increased, back to the original level (# 3 above).

7. Now we will put a hypoxic metabolic acidosis on top of the respiratory acidosis and see what is necessary to correct it. Again simulate the bronchial obstruction by bubbling CO₂ through your blood until the pH reaches a plateau. Record the change in pH as before.

8. In this case the decreased ventilation has also compromised oxygen delivery to the tissues. The patient has therefore become hypoxic resulting in increased glycolysis in the tissues and a consequent lactic acidosis (the Pasteur Effect). Continue bubbling CO₂ through your sample, but simulate the lactic acidosis by adding 200µl of the 0.1 M lactic acid solution to your sample, and keep recording the pH.

9. Simulate removing the obstruction and bubble air through the buffer as before. Record the change in pH every minute until the pH has reached a plateau. You will notice that the pH has not returned to "normal", this is because of the metabolic acidosis cannot be corrected by correcting the concentration of dissolved CO₂. The metabolic acidosis could be corrected by taking the pCO₂ lower than normal (a respiratory alkalotic compensation for the metabolic acidosis). This can happen in real-life situations, but we cannot do this in this experiment as our "normal" blood has a minimal concentration of
dissolved CO₂ to begin with. But a metabolic acidosis can also be corrected by administering alkali, so we will do this instead. In a real-life situation bicarbonate would be added, but since this is a buffering species (i.e. it can accept and donate H⁺) the effect of adding bicarbonate is complex. We will simply add the same number of moles of alkali (as acid) in the form of KOH.

10. Add 200μl of the 0.1 M KOH solution and record the pH.
LAB REPORT

Answer these questions.

1. Graph the changes in pH against time and explain what is happening in terms of equations (2) and (3).
2. Why are so many processes in the cell pH-dependent?
3. What is a buffer, what is a pK, and what are the implications of the pK for buffering?
RESPIRATION MODULE
Anatomy Laboratory

Wednesday, Week 12

The upper respiratory tract
Material used in this Laboratory:

Two sets of four tables

STATION 1: Nasal Cavity - Skulls- Half Skulls - Plastinated Heads hemisected

STATION 2: Para nasal Sinuses – Skulls - Radiographs

STATION 3: The Pharynx – isolated pharynx, pharynx in situ (from behind and in front)

STATION 4: Internal Larynx - Larynx Models - Larynx Bucket
Read and study these notes and use your textbook to become familiar with the contents BEFORE you come to the lab.

Outcomes

1. The student will be able to identify the major parts of the upper respiratory tract
2. The student will be able to describe parts, structure and function of the pharynx
3. The student will be able to identify the parts of the nasal cavity and paranasal sinuses, their nerve supplies and functions.
4. The student will be able to outline the structure and functions of the larynx.
5. The student will be able to recall the course of the recurrent laryngeal nerves, understand their function and the structures that can impinge on either the right or left recurrent laryngeal nerve, and recognise the symptoms of such impingement.
Station 1. THE NASAL CAVITY

Look at the nasal region on the skulls and half skulls provided. BE VERY CAREFUL because the bones around the nasal cavity and orbits are very thin. Skulls should be held with two hands and supported by the cranial base or with a finger in the foramen magnum. (Be aware that some of the skulls in the department have been previously damaged and have been repaired with plaster/filler)
If there is a skull in good condition available, hold the skull up to the light and see the translucent bone.

Identify the parts that surround the nasal cavity:
Anteriorly  nasal bones, maxillae, lachrymal, anterior nasal spine
Superiorly  frontal bone, ethmoid bone, cribriform plate crista galli
Laterally    Maxilla, inferior concha, ethmoid bone, orbit, palatine bone
Inferiorly  Palate, maxilla and palatine
Posteriorly Sphenoid body, Choana, pterygoid plates, vomer

What forms the nasal septum

Examine the specimens and models of the nasal cavity
Identify the nasal conchae what bone is each a part of:
Superior
Middle
Inferior

Identify the three recesses defined by the conchae and name the structures that open into each.
Sphenoethmoidal recess
Superior meatus
Middle meatus
Inferior meatus
Fig. 225. The lateral wall of the right nasal cavity; the conchae have been partially removed.
**Station 2. PARANASAL SINUSES**

Use the skulls and the radiographs to identify the paranasal sinuses – look at more than one to appreciate their variability

1. Maxillary sinus – note it forms the floor of the orbit and that the roots of the molar teeth project into the floor of the sinus.
   
   Why would dentists need to be careful in this area ________________

2. Frontal sinus – note that it lies in the forehead and also extends into the roof of the orbit.

3. Ethmoid air cells – These lie in the narrow space between the nasal cavity and the orbit, the bone here is extremely thin (Lamina papyracea).

4. Sphenoid sinus – Is in the centre of the skull – can you name two important structures that lie directly above it ____________________

What tissue lines the paranasal sinuses ____________________

The function of paranasal sinuses is still a mystery – outline some theories for their function ____________________

Compare the osteology to the radiographs provided.
Station 3. THE PHARYNX

Examine the specimens and models of the pharynx. The main function of the pharynx is swallowing and this will be dealt with in detail during the Digestive system module next year. The pharynx is also a respiratory passage and that aspect must be covered here.

Identify the 3 parts of the pharynx and answer the following questions

1. Nasopharynx
   What separates the nasopharynx from the oropharynx? __________________________
   What epithelium lines the nasopharynx __________________________
   Identify the opening of the Pharyngotympanic (Eustation or auditory) tube. Where does this tube lead to? __________________________
   What is the function of the auditory tube? __________________________

2. Oropharynx
   What sort of epithelium lines the oropharynx __________________________
   What structures mark the boundary between the oral cavity and the oropharynx
   Laterally __________________________
   Inferiorly __________________________
   Where would you look for the tonsils (Palatine tonsils)? __________________________

3. Laryngopharynx
   Lies behind the larynx and around the upper opening of the larynx.
   Identify the glossoepiglottic folds (1 median & 2 lateral) and the valleculae (spaces between the tongue and the epiglottis. Lateral to the laryngeal opening are the piriform recesses
Station 4. INTERNAL LARYNX

Use models and specimens to identify the parts of the laryngeal opening
- Epiglottis
- Aryepiglottic folds
- Arytenoid cartilages

Look into the larynx and observe the vestibular (false vocal) folds and the (true) vocal folds. The sheet of membrane forming the walls above the vestibular folds is called the quadrangular membrane.

The larynx has 3 functions:

1. Opening to allow air into and out of the lungs (abducting the vocal folds)
2. Closing to prevent food and drink from entering the lungs (this is done in two ways: (a) drawing down the epiglottis and (b) adducting the vocal folds)
3. Vocalisation (allowing air to pass between and cause the vocal folds to vibrate, and to alter the frequency of that vibration)
Intrinsic muscles of the larynx.
For each of the following muscles, use the figures here to help you to locate the muscle on specimens of the larynx. Also write down the action of the muscle and say with which of the major functions of the larynx it is involved:

Posterior crico-arytenoids

Lateral crico-arytenoids

Ary-epiglotticus

Thyro-epiglotticus

Inter-arytenoids (transverse and oblique)

Crico-thyroid

Thyro-arytenoids

Vocalis

What is the nerve supply of the intrinsic laryngeal muscles?

How might this help diagnose disease in the thorax?
RESPIRATION MODULE
Physiology Laboratory

Friday, Weeks 12 / 13

Case study I
CASE STUDY 1: INTERPRETATION OF LUNG FUNCTION TESTS AND BLOOD GASES

Objectives

- How obstructive and restrictive lung disease alter ventilation of the lung and gas exchange
- The concept of reversibility in obstructive lung disease
- The physiological basis of dynamic lung function tests such as FEV1, FVC, FEV1/FVC, FEV25-75% (same as MEFR25-75%), PEFR
- Be able to predict the type of disease present in the lung from the above recordings

In pairs you will work through 3 case studies using the SimBioSys simulator. The aims of the laboratory are to examine lung function data in the context of the physiological mechanisms concerned (for example, lung inflation, gas exchange, airways resistance). From basic concepts supplied in the lecture series you should then be able to reach informed conclusion as to the broad nature of disease in each case.

Each case presents a graphical display from the spirometer and also a flow volume plot for a force inspiration and expiration. On this display window you will see simulated values for dynamic (eg. PEFR) and static (FRC) tests. Blood gasses are also available if you click on the different tabs.

Get going

Select CASE 1 from CHAPTER 7.

Follow the instructions, firstly clicking on HERE to bring up the display window. Carefully read the patient information and then click where directed (HERE) to show 'live' breathing.

Press the button at the bottom of the display window to start a forced breath.

Examine the results for Dynamic and Static data. Also view the pO2 and pCO2 on the Metabolism page.

Together, discuss the meaning of the results and work out what you think is the problem. Try adding a bronchodilator to assist in your interpretation of the data.

Finally, click on the Diagnosis and Management link.

Repeat for CASE 2 and 3. At any time you can use the links to look at relevant basis physiology.
Assessment
There is no separate report or assessment for this case study. You will be provided with a hard copy of the Dynamic results (without bronchodilator, only) on leaving the class. Review this data during private study to fully understand what the lung function results show about the physiology of the lung and why the results differ from normal in the 3 disease situations.

This information may be required in the Laboratory and Theory Examinations.

Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC</td>
<td>Forced Vital Capacity</td>
</tr>
<tr>
<td>FEV1</td>
<td>Forced Expiratory Volume in 1 sec</td>
</tr>
<tr>
<td>FEV1/FVC</td>
<td>FEV1 as a percent of FVC</td>
</tr>
<tr>
<td>FEF 25-75</td>
<td>Forced Expiratory Flow over the mid region of the FVC</td>
</tr>
<tr>
<td>PEFR</td>
<td>Peak Expiratory Flow Rate</td>
</tr>
<tr>
<td>TLC</td>
<td>Total Lung Capacity</td>
</tr>
<tr>
<td>FRC</td>
<td>Functional Residual Capacity</td>
</tr>
<tr>
<td>RV</td>
<td>Residual Volume</td>
</tr>
<tr>
<td>Raw</td>
<td>Airways resistance (cmH2O/ml/min)</td>
</tr>
<tr>
<td>DLCO</td>
<td>Diffusing Capacity for Carbon Monoxide (ml/min/mmHg)</td>
</tr>
</tbody>
</table>
RESPIRATION MODULE
Physiology Laboratory

Friday, Weeks 12 / 13

Sleep Clinic
West Australian Sleep Disorders Research Institute
Queen Elizabeth II Medical Centre
Practical Class in Physiology of Sleep and Respiration

West Australian Sleep Disorders Research Institute
Queen Elizabeth II Medical Centre
Year I Medical

Learning Objectives
To understand:
1. Normal sleep architecture (the stages of sleep and their organization)
2. The physiological changes of sleep that affect respiration
3. How sleep and breathing during sleep can be monitored
4. The pathophysiology of obstructive sleep apnoea

Reading material
Material to be provided at tutorial.

Venue
Assemble in 8th floor Function Room, G Block (northwestern corner of building), Sir Charles Gairdner Hospital, for introductory lecture; then to Respiratory Sleep Disorders Clinic, Department of Pulmonary Physiology, on the 5th floor, for small group tutorials.

Duration
2 hours

Programme
1. Introductory Lecture (30 mins):
   “The spectrum of sleep-related breathing disorders” (Dr D R Hillman)

2. Small group tutorials (class divided into 5 groups) to cover 5 topics (15 mins each):
   2.1 Data acquisition
   Demonstration of methods used to acquire data relating to sleep and breathing (electroencephalography, electrooculography, submental electromyogram, oronasal airflow, chest wall motion, pulse oximetry, partial pressure of carbon dioxide (PCO₂).

   2.2 Obstructive sleep apnoea before and after treatment
   Representative data on computer workstation for discussion:
   respiratory parameters
   sleep hypnogram
   sleep stages and their influence on breathing

   2.3 Sleep hypoventilation before and after treatment
   Representative data on computer workstation for discussion:
respiratory parameters
sleep hypnogram
sleep stages and their influence on breathing

2.4 Cheyne Stokes Respiration, Multiple Sleep Latency Test, Narcolepsy
Representative data on computer workstation for discussion:
respiratory parameters

2.5 Treatment of Breathing Disorders of Sleep
Continuous positive airway pressure (CPAP) to treat
obstructive sleep apnoea
Dental devices to treat sleep apnoea
Non-invasive ventilation (NIV) to treat sleep hypoventilation
A pulse oximeter is provided to demonstrate measurement of
arterial oxygen saturation.

Assessment
This material will be assessed in the practical exam.
RESPIRATION MODULE
Physiology Laboratory

Available Online, Week 13

Case study II
CASE STUDY II: INTERPRETATION OF BLOOD GAS MEASUREMENTS - causes of hypoxemia

Objectives and background:

- Know normal values of alveolar (A), arterial (a) and venous (v) blood gases
- Use of alveolar gas equation to predict alveolar pO$_2$
- How V/Q mismatch and hypoventilation cause hypoxemia
- How to distinguish between them from the A-a pO$_2$ difference

It is often necessary, clinically, to distinguish between the main causes of severe hypoxemia. Two options are hypoventilation (e.g. caused by central depression, i.e. narcotic overdose) and V/A ratio mismatch. The latter can be as a result of severe asthma or emphysema.

Cardinal features of hypoventilation are a large increase in alveolar pCO$_2$ (PA$_{CO_2}$) and decreased alveolar pO$_2$ (PAO$_2$). What would you expect to happen to the difference between the alveolar pO$_2$ and the pO$_2$ of arterial blood, A-a pO$_2$ difference? (remember that the normal A-a pO$_2$ difference is only 5-10 mmHg and that this is mainly due to a shunting of blood past alveoli so that the blood does not come into contact with fresh air). What happens to the A-a pO$_2$ difference in V/Q mismatch (see Lecture on Hypoxemia)?

To distinguish between hypoxemia caused by hypoventilation and hypoxemia caused by V/Q mismatch we measure the A-a pO$_2$ difference.

This can be done at the bedside from arterial blood gases (paO$_2$ and paCO$_2$) and using the alveolar gas equation (dealt with in lectures).

Assessment

There is an on-line assessment exercise. A table similar to the one below should be completed on the web, by the date to be advised. It will be marked and goes towards the continuous assessment of the unit.

Calculations needed for the table

1) The alveolar gas equation includes the respiratory exchange ratio, or respiratory quotient, which under resting conditions is close to 0.8. Make sure you know what R is and how it depends on the food you are burning for energy.

2) Make sure you know how to calculate partial pressures for room air and for warmed moistened air passing towards the alveoli.

3) Use the alveolar gas equation to calculate the PAO$_2$ in the examples below (NB. For this calculation assume that PACO$_2$ and paCO$_2$ are the same).

4) Work out the A-a pO$_2$ difference.
5) Based on your findings, which patients probably had V/Q mismatch and which hypoventilation?

<table>
<thead>
<tr>
<th>patient</th>
<th>$\text{paO}_2$ mmHg</th>
<th>$\text{paCO}_2$ mmHg</th>
<th>inspired gas</th>
<th>Calculated $\text{pAO}_2$ mmHg</th>
<th>A-a $\text{pO}_2$ difference</th>
<th>Hypoventilation or V/Q mismatch</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>95</td>
<td>40</td>
<td>air</td>
<td>100</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
<td>air</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**APPENDIX I**

**NUMBERS IN PHYSIOLOGY**

The following figures give mean values for healthy young adults. Variation of up to 20% from the mean is usually within normal limits.

**FIGURES MARKED WITH AN ASTERISK SHOULD BE MEMORISED. YOU WILL BE TESTED ON THESE VALUES AT SOME STAGE IN YOUR PHYSIOLOGY COURSE,** and they will be a great help when you have to calculate some of them in exam questions, to give you an idea whether you are in the right ballpark with your answer. The remaining figures have been inserted as an aid to the understanding of physiology or because of their important applications.

**BLOOD, PLASMA, WATER, ELECTROLYTES**

<table>
<thead>
<tr>
<th>Description</th>
<th>Value</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Red blood cell count</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- adult female</td>
<td>5.0 x 10^{12}/l</td>
<td></td>
</tr>
<tr>
<td>- adult male</td>
<td>5.5 x 10^{12}/l</td>
<td></td>
</tr>
<tr>
<td><em>Life span of erythrocyte</em></td>
<td>120 days</td>
<td></td>
</tr>
<tr>
<td><em>Diameter of erythrocyte (wet film)</em></td>
<td>8 micrometres (µm)</td>
<td></td>
</tr>
<tr>
<td><em>Haemoglobin content</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- adult female</td>
<td>135 g/l blood</td>
<td></td>
</tr>
<tr>
<td>- adult male</td>
<td>150 g/l blood</td>
<td></td>
</tr>
<tr>
<td><em>White blood cell count in adult</em></td>
<td>4-10 x 10^9/l</td>
<td></td>
</tr>
<tr>
<td>(60% neutrophil polymorphs.)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Platelet count</em></td>
<td>250 x 10^9/l (150-400)</td>
<td></td>
</tr>
<tr>
<td><em>Iron content of body</em></td>
<td>4.5g (2/3 as Hb)</td>
<td></td>
</tr>
<tr>
<td><em>Iron requirement daily</em></td>
<td>14 mg (1-3 mg absorbed)</td>
<td></td>
</tr>
<tr>
<td><em>Serum iron</em></td>
<td>20 µmol/l (13-22)</td>
<td></td>
</tr>
<tr>
<td><em>Bleeding time (Ivy)</em></td>
<td>Up to 5 min</td>
<td></td>
</tr>
<tr>
<td><em>Whole blood clotting time.</em></td>
<td>Variable, ≈ 5-11 min</td>
<td></td>
</tr>
<tr>
<td><em>One stage “prothrombin time” (Quick)</em></td>
<td>Variable, ≈ 12-14 sec</td>
<td></td>
</tr>
<tr>
<td>(or 1.8 - 3 x control)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Mean corpuscular haemoglobin concentration (MCHC)</em></td>
<td>340g/l (320-350)</td>
<td></td>
</tr>
<tr>
<td><em>Mean corpuscular volume (MCV)</em></td>
<td>90 fl (82-97)</td>
<td></td>
</tr>
<tr>
<td><em>Packed cell volume</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- female</td>
<td>42%</td>
<td></td>
</tr>
<tr>
<td>(haematocrit)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- male</td>
<td>45% (43-49%)</td>
<td></td>
</tr>
<tr>
<td><em>Blood oxygen capacity</em></td>
<td>200 ml/l at STP</td>
<td></td>
</tr>
</tbody>
</table>

*1 g haemoglobin combines with 1.36 ml oxygen at STP*

Plasma CO₂ content

480-520 ml/l at STP

(23 - 27 mmol/l)

Ratio plasma bicarbonate / dissolved CO₂

(24 / 1.2) mmoles =20

pK value of CO₂ - bicarb. system in plasma

6.1
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH of plasma</td>
<td>7.35 - 7.45</td>
</tr>
<tr>
<td>Body water (male)</td>
<td>60% of total body wt.</td>
</tr>
<tr>
<td>Body water (female)</td>
<td>50% of total body wt.</td>
</tr>
<tr>
<td>NOTE:</td>
<td>Body water varies with fat content, fat people have lower body water</td>
</tr>
<tr>
<td>Body water (male &amp; female)</td>
<td>73% LEAN body mass</td>
</tr>
<tr>
<td>Cellular water</td>
<td>~ 25 litres (70kg man)</td>
</tr>
<tr>
<td>Plasma water</td>
<td>~ 3 litres (70kg man)</td>
</tr>
<tr>
<td>Blood content (lean person)</td>
<td>8% of weight</td>
</tr>
<tr>
<td></td>
<td>(~ 5 litres, 65kg man)</td>
</tr>
<tr>
<td>Total osmotic pressure of plasma</td>
<td>$7 \times 10^2$ k Pa</td>
</tr>
<tr>
<td></td>
<td>(7 atm., 5320 mm Hg)</td>
</tr>
<tr>
<td>Colloid osmotic pressure of plasma</td>
<td>3.3 k Pa (25 mm Hg)</td>
</tr>
<tr>
<td>Plasma osmolality</td>
<td>280 - 300 mosmol/litre</td>
</tr>
<tr>
<td>Blood specific gravity</td>
<td>1.060 or less</td>
</tr>
<tr>
<td>Plasma specific gravity</td>
<td>1.030 or less</td>
</tr>
<tr>
<td>Specific gravity of plasma ultra-filtrate</td>
<td>1.010</td>
</tr>
<tr>
<td>Freezing point depression of plasma</td>
<td>0.56°C</td>
</tr>
<tr>
<td>Plasma sodium</td>
<td>140 mmol/l</td>
</tr>
<tr>
<td>Plasma potassium</td>
<td>4 mmol/l</td>
</tr>
<tr>
<td>Plasma calcium</td>
<td>2.5 mmol/l or 10 mg/dl</td>
</tr>
<tr>
<td></td>
<td>(half of this is ionised)</td>
</tr>
<tr>
<td>Plasma chloride</td>
<td>105 mM</td>
</tr>
<tr>
<td>Plasma bicarbonate</td>
<td>25 mmol/l</td>
</tr>
<tr>
<td>Plasma phosphate</td>
<td>1 mmol/l</td>
</tr>
<tr>
<td>Physiological saline</td>
<td>0.85% NaCl or 0.15 M</td>
</tr>
<tr>
<td>Body sodium</td>
<td>4,000 mmol</td>
</tr>
<tr>
<td></td>
<td>(70% rapidly exchangeable)</td>
</tr>
<tr>
<td>Body potassium</td>
<td>3,400 mmol</td>
</tr>
<tr>
<td></td>
<td>(90% rapidly exchangeable)</td>
</tr>
<tr>
<td>Plasma proteins</td>
<td>70g/l</td>
</tr>
<tr>
<td>Plasma glucose (fasting)</td>
<td>4 mmol/l (80 mg/100 ml)</td>
</tr>
<tr>
<td>Plasma urea</td>
<td>4 mmol/l (25 mg/100 ml)</td>
</tr>
<tr>
<td>Blood sedimentation rate</td>
<td>2 - 7 mm per hour</td>
</tr>
</tbody>
</table>
Note 1: **Electrolytes** can be expressed as above in terms of molar solutions (eg. $23\text{ [Na]} + 35.5\text{ [Cl]}$, a total of 58.5 g NaCl per litre solution gives 1M); or as molal solutions eg. $l\text{ gm mol. wt/kg water}$. (An older system also presents electrolytes as equivalents, eg. mEq/litre. For univalent ions 1 mmol equals 1 mEq, for divalent ions 1 mmol equals 2 mEq. We try not to use this system any more but it occasionally slips in)

Note 2: **Osmotic pressure**: The osmotic pressure of a 1M solution of a non-electrolyte is one osmole/litre. Osmotic pressure is measured clinically by measuring freezing point depression. An ideal 1M solution will depress freezing point by $1.86^\circ\text{C}$, so plasma should have a freezing point of about $1.86 \times 300/1000$ (0.56)$^\circ\text{C}$ below zero, a change which modern osmometers can measure to better than 1% accuracy. Osmotic pressure is expressed as mosmol/litre. For strong electrolytes (eg. NaCl) the osmotic pressure will usually be less than calculated for the sum of mmoles/litre of the ions, owing to inter-ionic attraction. For instance you have to dissolve 9.421 g of NaCl in 1 litre of water to get a 300 mosmol/litre solution but this is 0.161 moles/litre or 322 mmoles/litre total.

There is only a small difference between osmolality and osmolarity (osmolality is osmoles/kg water, osmolarity is osmoles/litre solution) but it is best to stick to osmolality in medicine since it is not affected by the protein content of the plasma.

Note 3: **Clinical values** are very often reported as $Xm/100\text{ ml (dl)}$, rather than $/\text{litre}$. Watch out for this and be prepared to make the appropriate adjustment in any calculations. We try to use S.I. in everything (except B.P.) but some of the other values are still around.
RESPIRATION
Mean Values

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total lung volume</td>
<td>6000 ml</td>
</tr>
<tr>
<td>Vital capacity</td>
<td>4500 ml</td>
</tr>
<tr>
<td>Residual volume (after max. expiration)</td>
<td>1500 ml</td>
</tr>
<tr>
<td>Functional residual capacity</td>
<td>2300 ml</td>
</tr>
<tr>
<td>Tidal volume</td>
<td>400-500 ml</td>
</tr>
<tr>
<td>Anatomic dead space</td>
<td>150 ml</td>
</tr>
</tbody>
</table>

Forced expired volume (FEV₁) > 80% of vital capacity in 1 sec

Maximum breathing capacity 150 litre/min

Pulmonary ventilation (VE) or respiratory minute volume (RMV) 7 5 litre/min

Alveolar ventilation 5.25 litre/min

Oxygen consumption at rest 250 ml/min

Haemoglobin in blood 15g/100 ml

Haemoglobin (Hb) - oxygen carriage 1g carries 1.39 ml O₂

O₂ content of Hb at 100 mm PaO₂ 20.85 ml O₂/100ml blood

O₂ content of Hb at 100 mm PaO₂ 20 ml O₂/100ml blood

Basal metabolic rate 168 k Joules/m²/h

Surface area of average man ≈ 1.7 m²

Partial Pressure of water at 37°C 47 mmHg

*PARTIAL PRESSURE (mmHg)*

<table>
<thead>
<tr>
<th></th>
<th>PO₂</th>
<th>PCO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atmosphere (dry)</td>
<td>159</td>
<td>0</td>
</tr>
<tr>
<td>Inspired gas (saturated)</td>
<td>149</td>
<td>0</td>
</tr>
<tr>
<td>Alveolar gas (saturated)</td>
<td>100</td>
<td>40</td>
</tr>
<tr>
<td>Arterial blood</td>
<td>90-98</td>
<td>40</td>
</tr>
<tr>
<td>Venous blood</td>
<td>40</td>
<td>46</td>
</tr>
<tr>
<td>tissues</td>
<td>&lt;30</td>
<td>&gt;50</td>
</tr>
</tbody>
</table>
KIDNEY AND URINE

Blood flow

1250 ml / min

*Plasma flow (p-amino-hippurate, PAH, clearance) 650 ml / min

*Glomerular filtration rate (inulin, creatinine clearance) 120 ml / min

*Filtration fraction 120 ÷ 650 = 0.18

Urine (very variable) ~ 1 ml / min

Non-urinary water loss (very variable) ~ 1 litre / day

Urine

specific gravity 1.001 - 1.032

*osmolarity 50-1100 mosmol/litre

*pH 4.8 - 8.0

Daily urinary output (mixed diet: variable)

urea approx. 30 g
ammonia approx. 0.7 g
uric acid approx. 0.7 g
creatine approx. 1 g
NaCl approx. 100 mmol
K approx. 80 mmol
Titratable acidity (pH 7, phenol red) 20 mmoles/day
Ammonia 50-75 mmol daily (average diet)

Kidney function tests

Urea clearance 75 ml / min
Tubular maximum glucose 350 mg (2mmoles)/min
Tubular maximum, PAH. 75 mg (0.4mmoles)/min

Sweat

NaCl 25-50 mmol/litre (always hypotonic)
CIRCULATION

*Cardiac output (rest) 5 litres/min
*Cardiac output (exercise) up to 30 litres/min
*Stroke volume 70 ml
*Systolic/diastolic pressure 120/80 (mm Hg)
*Pulse pressure (120 minus 80) 40 (mm Hg)
*Pulmonary artery blood pressure 25/8 (mm Hg)
*Central venous pressure 2-5 (mm Hg)
(mean R. atrial pressure)

NB 1 mm Hg = 14 mm H2O

NERVOUS SYSTEM AND SPECIAL SENSES

Conductance time in
  A fibres approx. 100 m per sec
  C fibres 2 m per sec

C.S.F.
  Volume 150 ml
  Cells 5 per mm3
  Protein 200 mg/l

*CSF Pressure 100-200 mm H2O
  (lateral position)

Eyes
  Total refractive power 60 Dioptres
  Near point (young adult) 10 cm
  Intraocular pressure 17 mm Hg

Hearing
  Average limits in young adult : 20-20,000 cycles per second (cps or Hertz)
  The threshold is lowest (ie hear best) between 1500-3000 cycles per second
DIET

Calorific value: Note 1 kilocalorie (Kcal) = 4.2 kJoules

1 g protein 17 kJ (4.1 Kcal)
1 g carbohydrate 17 kJ (4.1 Kcal)
1 g fat 39 kJ (9.3 Kcal)
small loaf (350g) 4186 kJ (1000 Kcal)

Daily calorific requirement

*basal (resting) 7500 kJ (1800 Kcal)
*labourer 17000 kJ (4000 Kcal)

DIGESTION

Gastric juice pH 1 approx

*Digestive fluids water secreted approx. 10 litres/day

GUIDE TO S.I. UNITS

The International Organisation of Standardisation has attempted to standardise the symbols used in scientific literature and at the same time devise a system of metric units that would be acceptable to all branches of science.

SI units consist of base units with decimal multiples and submultiples and some supplemental units.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Name of Unit</th>
<th>Symbol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length</td>
<td>Metre</td>
<td>m</td>
</tr>
<tr>
<td>Mass</td>
<td>Kilogram</td>
<td>kg</td>
</tr>
<tr>
<td>Time</td>
<td>Second</td>
<td>s</td>
</tr>
<tr>
<td>Amount of substance</td>
<td>Mole</td>
<td>mol</td>
</tr>
<tr>
<td>Pressure</td>
<td>Pascal</td>
<td>Pa</td>
</tr>
<tr>
<td>Volume (see note 4 below)</td>
<td>litre</td>
<td>l</td>
</tr>
</tbody>
</table>
NOTES:

1) One Pascal (Pa) is defined as a pressure of 1 Newton/sq. metre.
2) The mass concentration (weight of substance per unit volume) is expressed in g/l or mg/l etc.
3) The substance concentration (moles of a substance per unit volume) is expressed in mol/l or mmol/l etc.
4) The unit of volume should rationally be the cubic metre, but this is a very large unit and as the litre is a well-established unit and 1 cubic metre = 1000 litre, it is still used.
5) The term 100 ml will be discontinued and replaced by the decilitre (abbreviated dl).

**Prefixes for SI Units**

\[
\begin{array}{ccc}
10^{12} & \text{tera (T)} & 10^{-18} \\
10^9 & \text{giga (G)} & 10^{-15} \\
10^6 & \text{mega (M)} & 10^{-12} \\
10^3 & \text{kilo (k)} & 10^{-9} \\
10^2 & \text{hecto (h)} & 10^{-6} \\
10^1 & \text{deca (da, dk)} & 10^{-3} \\
& & 10^{-2} \\
& & 10^{-1} \\
& & \text{deci (d)}
\end{array}
\]

Note that d means deci (1/10) not deca (100). (There is almost unlimited possibility for error here!).

**Common conversion factors**

<table>
<thead>
<tr>
<th>Conversion</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mmHg = 133.32 Pa</td>
<td>1 k Pa = 7.5006 mmHg</td>
</tr>
<tr>
<td>1 atmosphere = 1.013 x 10^5 Pa</td>
<td>1 cal = 4.184 J</td>
</tr>
</tbody>
</table>

**Conversion of mass concentration to molar concentration**

eg:

urea at 40 mg/100 ml

Molecular weight of urea = 60

\[
\text{Concentration} = \frac{400 \text{ mg/l}}{60} = 6.6 \text{ mmol/l}
\]
# APPENDIX II

## SYMBOLS USED IN RESPIRATORY PHYSIOLOGY

### PRIMARY

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>Concentration of gas in blood</td>
</tr>
<tr>
<td>F</td>
<td>Fractional concentration in dry gas</td>
</tr>
<tr>
<td>P</td>
<td>Pressure or partial pressure</td>
</tr>
<tr>
<td>Q</td>
<td>Volume of blood</td>
</tr>
<tr>
<td>Q̇</td>
<td>Volume of blood per unit time</td>
</tr>
<tr>
<td>R</td>
<td>Respiratory exchange ratio</td>
</tr>
<tr>
<td>S</td>
<td>Saturation of hemoglobin with $O_2$</td>
</tr>
<tr>
<td>V</td>
<td>Volume of gas</td>
</tr>
<tr>
<td>V̇</td>
<td>Volume of gas per unit time</td>
</tr>
</tbody>
</table>

### SECONDARY SYMBOLS FOR GAS PHASE

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Alveolar</td>
</tr>
<tr>
<td>B</td>
<td>Barometric</td>
</tr>
<tr>
<td>D</td>
<td>Dead space</td>
</tr>
<tr>
<td>E</td>
<td>Expired</td>
</tr>
<tr>
<td>I</td>
<td>Inspired</td>
</tr>
<tr>
<td>L</td>
<td>Lung</td>
</tr>
<tr>
<td>T</td>
<td>Tidal</td>
</tr>
</tbody>
</table>

### SECONDARY SYMBOLS FOR BLOOD PHASE

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>a</td>
<td>arterial</td>
</tr>
<tr>
<td>c'</td>
<td>capillary</td>
</tr>
<tr>
<td>c</td>
<td>end-capillary</td>
</tr>
<tr>
<td>i</td>
<td>ideal</td>
</tr>
<tr>
<td>v</td>
<td>venous</td>
</tr>
<tr>
<td>v̇</td>
<td>mixed venous</td>
</tr>
</tbody>
</table>

### EXAMPLES

- $O_2$ concentration in arterial blood $C_a{O_2}$
- Fractional concentration of $N_2$ in expired gas $F_{E{N_2}}$
- Partial pressure of $O_2$ in mixed venous blood $P_{v{O_2}}$
- Tidal Volume $VT$
- Volume Expired per minute $V̇_E$ (or inspired $V̇_I$); used to be called "Respiratory Minute Volume" (RMV)